

SYNTHETIC ELECTROCARDIOLOGY

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SYNTHETIC ELECTROCARDIOLOGY

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The author, on the basis of his own experience in a modern cardiological clinic using electrophysiological and biophysical methods of heart study, approaches these methods from a single viewpoint. This is accomplished by passing from the analytic examination of each investigative method to a synthesis of all these methods. In this work, the questions pertaining to modern clinical electrocardiology are investigated on the basis of a synthetic principle. This monograph is, in fact, a first attempt at defining clinical electrocardiology as a separate branch of cardiology. The book is intended for electrocardiologists, practicing physicians, surgeons, and physiologists working in the domain of clinical and experimental cardiology. There is a concise English annotation at the end of the book.

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author

FOREWORD

In the rich arsenal of diagnostic techniques embodied in modern cardiology, one of the most important places is occupied by electrocardiography which, in spite of its comparatively short history, has already achieved tremendous success. At the present time, this method is widely used in medical practice, and the problems connected with its theoretical foundations are quite thoroughly developed. During recent years, singular achievements have been made in the application of the new electrocardiographics and in the treatment of the electrical forces in the mechanism of the heart on the basis of the principles of vector analysis.

On the other hand, a modern cardiac specialist is not able to confine himself only to an investigation of the electrical activity of the heart. To obtain a detailed and profound understanding of the activity of the heart, it is also necessary to investigate the mechanical activity of the heart and thus to fill the gap which remained following the investigation of the electrical manifestations of cardiac contractions.

On the basis of these considerations, the author of this book, Z. L. Dolabchyan, Candidate of Med. Sc., consolidates a series of modern electrocardiological methods into a general synthetic whole. He bases his clinical investigation of electrical cardiac activity on the methods of electrovectorial and vectorial electrocardiography, while he bases his studies of the mechanical activity of the heart on such methods as ballistocardiography, phonocardiography and the combined polygraphic investigation which, taken together, create the possibility of obtaining an idea not only of the con-

/Numbers in the margin indicate pagination in the original foreign text.

tractile ability of the heart, but also of the auditory phenomena and the phases of cardiac contraction. On the basis of his practical experience, the author concludes that the approach to electrocardiological methods must be begun from the synthetic viewpoint: passing from the analytical investigation of each separate method to the general synthesis of data obtained by all of these methods.

Z. L. Dolabchyan's book is based on the author's experiences over a 10-year period. The language in which this book is written is of a high scientific level and it makes interesting reading. The guide to synthetic electrocardiography offered by the author is a new introduction into the science of modern electrocardiology and is of definite scientific and practical significance. While offering this, to a certain extent original, guide to the analysis of theoretical and practical problems in electrocardiology, the author also recognizes the necessity of creating laboratories of synthetic electrocardiology at large clinics and hospitals. /6

Z. L. Dolabchyan's work is dedicated to an urgent and important problem of modern medicine. The appearance of such a monograph in the contemporary literature on cardiology may be of great scientific and practical aid to physicians and scientific personnel working in the area of clinical and experimental cardiology.

Active Member of the Academy of Medical Sciences of
the U.S.S.R. and of the Academy of Sciences of the
Armenian S.S.R.

Professor L. A. Oganesyan

INTRODUCTION

Sixty years have passed since the day when Einthoven introduced his simple electrocardiograph which was destined to play a major role in the development and shaping of cardiology as a separate branch of medicine. The practical and theoretical problems investigated by Einthoven and his collaborators opened a wide range of possibilities for other investigators and, by the twenties, electrocardiography had assumed its full scientific role due to the effort of scientists at home and abroad. At present, the science of electrocardiography has reached a high level of development. It is now widely used in clinics in conjunction with new electrophysiological methods of heart study. If the cardiology of today is of such outstanding importance, if it has become possible to penetrate into the finest recesses of the heart's mechanism, if the hand of the surgeon now has the temerity to cut into this last forbidden organ, all this has become a reality due principally to the rapid development of electrocardiology, which is the alphabet and the cornerstone of cardiology as we now have it. /7

Such basic methods of modern cardiology as electro-, vectorial-, ballisto-, phono-, and polycardiography are now being widely used and scientifically investigated in the electrocardiological laboratory of the Institute of Cardiology and Cardiac Surgery in the Academy of Sciences of the Armenian S.S.R. In the process of our investigations, we have established that each of these methods has its limitations, i.e. that each of them taken separately is useful in studying only individual heart mechanisms and can supply answers only to a limited number of questions. On the basis of our experience, we have arrived at a firm conclusion that it is necessary to approach these methods from a single synthetic point of view: to pass from the analytical study of data obtained by means of each separate method to the general synthesis of data obtained by all methods. Moreover, we have found that such a synthesis must be done by one single specialist working according to a definite preconceived plan.

With such a formulation of the problem, the significance and the specific gravity of electrocardiological research becomes of considerably greater importance in the complex of cardiological methods. In this connection, we feel that it is not entirely appropriate when specialists in these methods, working in the same clinical institution, perform their work in almost complete isolation from one another. When, for instance, the specialist in electrocardiography is isolated from the specialist in ballistocardiography, or when the specialist in phonocardiography is almost ignorant of the work of the specialist in vectorial cardiography. Such arrangements are faulty because, under these conditions, electrocardiological analysis is always incomplete, and a large number of questions remain unanswered. /8

This basically new concept of clinical electrophysiology and biophysics we have named "Synthetic Electrocardiology." We have unified all previously mentioned methods into a single term of "electrocardiology", certainly not from the etymological standpoint, but on the basis of the substance of this technical term and its modern meaning. It is possible, of course, to include

other methods in the scope of this research, but as discussed in the second chapter of the first part of this book, it is already quite difficult for a specialist to master all of these complex methods. We are certain that with a correct application of the proposed methods, an investigation of synthetic electrocardiology will result in a very rich and versatile conception of the functional and particularly the anatomical state of the heart.

It must be taken into consideration that this work is not a textbook. Therefore, we will not consider here any of the isolated practical and theoretical problems, or problems of a general clinical character. We will discuss only those syndromes or conditions which can be investigated by the proposed method, and we emphasize only those conditions which may have a significance in the proposed method. Thus, for example, the question of age or significance of respiration are discussed in detail in the section on ballistocardiography because these factors are of importance for this method. The question of heart position is discussed in the section on electrocardiography because of the role played by this method in the determination of different heart positions.

In the preparation of this text, we have adhered to international terminology while also giving the corresponding Russian nomenclature. General data were taken from domestic and foreign literature when these data were in agreement with our own ideas.

The illustrative material of the book was based on our own observations (all except Fig. 71). All schematic drawings were prepared by ourselves. The heart operations were performed in the surgical division of the Institute by Doctor of Medicine A. L. Mikayelyan.

While offering this course on synthetic electrocardiology and developing its practical and theoretical aspects, we stress the necessity of creating laboratories for synthetic electrocardiology in large clinics and hospitals. We do not intend to minimize the role of the narrow field specialists (scientific workers) in separate branches of electrocardiology. On the contrary, we think that these specialists are of great importance in the development of electrocardiology. Our science must be furthered by the combined effort of clinicians, physiologists, biophysicists, electrical engineers and other specialists. This is of special importance in our atomic era, where there is an urgent necessity for telemetric investigations of space flights of man and when the methods of cybernetics and electronic computers have found wide application in medicine. /9

I take this opportunity to express my deep gratitude to the editor of my book, my dear teacher - active member of the Academy of Sciences of the U.S.S.R., who is also a member of the Academy of Sciences of the Armenian S.S.R., - L. A. Oganesyanyan for his great help and fatherly approach, which I constantly felt from the very first days of my scientific medical career.

It is a pleasant duty to express my sincere gratitude to the Director of the Institute of Cardiology and Cardiac Surgery of the Academy of

Sciences of the Armenian S.S.R. - Assistant Prof. K. A. Kyandaryan, for the opportunity he gave me to carry out this work and for his constant interest in it.

Heartfelt thanks also go to all my co-workers in the electrocardiological laboratory and all the personnel of the Institute whose collaboration made possible the preparation of this work.

PART ONE

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PRINCIPLES OF SYNTHETIC ELECTROCARDIOLOGY

CHAPTER I

ELECTRICAL AND MECHANICAL ACTIVITY OF THE HEART

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I. INTRODUCTION

The development of clinical cardiology made an early transition from the period of anatomical, descriptive investigations to the new period of functional trends. From this standpoint, a clinic requires good methods for investigation of the functional condition of the heart. This fact assumes special significance in modern cardiology where there is a necessity of mastering the clinical pathophysiology of the mechanism of the heart and where the surgeon's scalpel must act freely within this last "virginal" organ.

The intricate complexity of cardiac activity is based on various biochemical and biophysical processes whose dynamic condition determines the functional state of the heart.

The cardiology of today has attained certain achievements in the realm of the study of the biochemistry of the myocardium under normal and pathological conditions (Kh. S. Koshtoyants and T. M. Turpayev, 1946; Hoeglin, 1959; Szent-Györgyi, 1959; Raab, 1959; Lenzi, 1959; S. Ye. Severin, 1961; M. Ye. Rayskina, 1962, etc.) This aspect of cardiology is, however, considerably less developed than the others, and P. D. Gorizontov (1959) is right when he refers to it as "a new departure" in cardiology. Unfortunately, the biochemistry of today may be only of indirect help to a clinical cardiologist, since an investigation of the metabolism of the human myocardium is still impossible "in vivo," although certain authors (Sutton and Sutton, 1960) have been able to perform a biopsy on a non-exposed heart, which may give some material for further biochemical investigations (we do not speak here of the biopsies performed during heart surgery).

The complicated biochemical and biophysical processes determining the mechanism of cardiac activity lead in the end to an appearance of two basic groups of phenomena: electrical and mechanical. Since both are but physical processes, these phenomena open great possibilities for clinical research, especially under the conditions of the modern development of science when tremendous achievements in radio-electronics have created all the prerequisites for improved equipment.

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In isolating the electrical and mechanical cardiac phenomena, we do not discern any great difference between them; in fact, they are so interdependent that, perhaps, it would be more correct to call them electromechanical phenomena. This, however, is only the theoretical or purely physiological side of the question. Our division of cardiac activity into two separate groups of phenomena is based on our daily clinical considerations, since each of these groups of phenomena requires an entirely different method of approach, as well as different equipment. For a clearer understanding of

these conditions, it is necessary to discuss the anatomical and physiological properties of the heart, i.e. having significance from the standpoint of clinical cardiology.

II. ANATOMIC FEATURES

In contrast to other parts of the vascular system, the heart is developed from two rudimentary beginnings: the endothelial tube and the so-called myoepicardial plate (A. A. Zavarzin and S. I. Shchelkunov, 1954). Formation of these colonies of cells in the cranial end of the embryo begins in the second week of embryonic life. The heart acquires its final form at the end of the second month of development. Of the three membranes in the heart structure, the middle one - the myocardium - is of the greatest interest from both the anatomical and functional points of view.

Histologically, myocardial fibers with their abundant sarcoplasm and their centrally located nuclei resemble smooth muscle, while their longitudinal arrangement and transversely striated structure (not as clearly defined as in a skeletal muscle) resemble skeletal muscles. But the myocardium differs from these types of muscular tissue by the fact that its fibers are connected with one another by peculiar bridges, imparting a syncytial structure to the myocardium. A transverse striated fiber of the myocardium is covered with a structureless membrane separating the specific ionic medium of the fiber from the surrounding medium. The chemical structure of cardiac muscle does not differ basically from that of skeletal muscle, although it has been suggested that cardiac muscle consists of a different type of myosin (Bourne, 1960).

From the functional point of view, it is possible to assume that the heart consists of two syncytial units: the upper unit common to both auricles and the lower unit common to both ventricles. With the exception of a small muscular bridge, these two groups are completely separated from one another by a fibrous ring having four openings: two auriculoventricular openings and two others for the expulsion of the main blood vessels (the aorta and the pulmonary artery.) The muscular fibers within these masses of myocardium have a complex arrangement. It is possible to discern the superficial fibers common to the right and left halves of the heart, and the deeply seated fibers characteristic of the individual heart chambers. /15

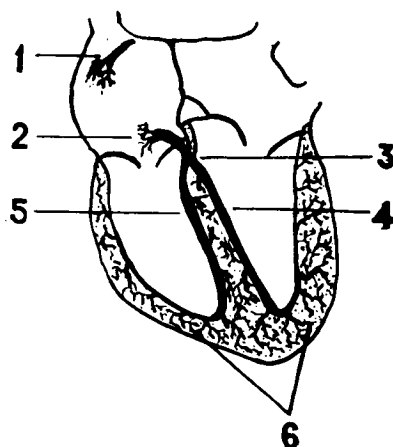
Normal cardiac ventricles can have different weights. They contain about 38 billion fibers, equally distributed between the two ventricles. On the basis of data obtained by Linzbach, Pendle (1959) writes that a normal heart has a uniform standard body of muscle whose proportions do not change during normal growth. However, the increase in the heart's efficiency with the development of the organism leads to a stretching of the muscle fibers and a relative increase in their surface.

This information pertains to the contractile part of the myocardium. The myocardium also has a second part, however - the conductive or modular part. Histological examinations of this tissue show that in the human body it has the same structure as the contractile part of the myocardium but is

richer in nuclei and sarcoplasm, poorer in myofibrils, and is often accompanied by ganglionic neurons. The muscular fibers of the modular tissue do not have contractile ability and are rather similar to the embryonic cells of the myocardium; they are not neuromuscular structures, as is assumed by certain authors, but represent a specialized type of cardiac muscle.

The modular tissue has a definite location in the mass of the myocardium (Fig. 1). From the anatomical point of view, it can be divided into auricular and ventricular parts, but from the functional standpoint it is more expedient to divide it into the sino-auricular and atrioventricular systems.

The anatomical structure of the sino-auricular node was described by Keit and Flack in 1906; this node is frequently called Keit-Flack's node. This node is located to the right of the place of entry of the upper vena cava into the right auricle, in the upper part of the crista terminalis. It starts subepicardially, then proceeds dorsocaudally and ends in the subendocardial layers. The length of the node is 20-25 mm, and its width is 2 mm. Schematically it can be divided into three parts: head-part, stem part, and the lower fork. Basically, the node consists of thin fibers which are parallel within the stem and fork parts of the node and are intertwined in the head part. There is no sharp demarcation between the node and the auricular muscles.



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Fig. 1. Scheme of the arrangement of the conductive or nodal part of the myocardium. 1-sino-auricular node; 2-atrioventricular node; 3-common stem of the bundle of His; 4-left leg of the bundle of His; 5-right leg of the bundle of His; 6-final ramifications of the bundle of His.

The atrioventricular system differs from the sino-auricular node in its more complicated structure. It starts from the atrioventricular node located above the opening of the venous sinus among the annular and partitional fibers of the right auricle, and proceeds along the partition dividing the auricle and ventricle. The histological system has the same structure as the sino-auricular node. The shape of the atrioventricular node resembles a mushroom formation with a length of 5 mm and width of 23 mm. Some authors call it simply "the node," while others call it the Aschoff-Tawara node in honor of

the authors who described it for the first time.

In its lower part, the node merges into the common stem of the atrioventricular system and bundle of His which extends towards the apex of the heart, i.e. forward, to the left and in the caudal direction. The bundle has a length of 10 mm, a width of 3 mm and divides itself, at the level of the beginning of the muscular part of the interventricular partition, into two bundle branches, one for the left and one for the right ventricle. The right bundle branch of the bundle of His seems to be a continuation of the common stem; it is located under the endocardium and soon reaches, without ramification, the forward papillary muscle. The left bundle branch is considerably shorter; it passes into the left side of the partition and very soon goes into considerable ramifications. Both bundle branches end in the subendocardial layer of the ventricles in a final network of Purkinje cells. These cells are scattered in the mass of the myocardium so that their quantity decreases with increasing distance from the endocardium and with the approach to the subepicardial layers. The typical large Purkinje cells which are encountered in certain animals are never found in humans.

In addition to describing the localization of the conductive system, certain authors have described other aspects of the arrangement of elements of the nodal tissue. Thus, they point out the presence of separate islets of nodal tissue within the walls of the auricles and ventricles; they also describe the left sino-auricular node situated within the interauricular partition and intended for the functioning of the left auricle. Separate bundles uniting the two auricles have also been described. Kent and other authors point out the fibers uniting the auricle with the ventricles. Gresham (1957) finds additional concentrations within the right auricle and also in /17 the right ventricle. These findings, together with some others discussed in the literature, are not generally accepted; therefore, in our work, we adhere to the classical description of the conductive system.

III. PHYSIOLOGICAL FEATURES

The physiology of the heart is characterized by four (according to certain authors, by five) basic functions of the myocardium: automatism, excitability, conductivity, and contractility.

1. Automatism

The heart has the ability to produce its own impulses without aid from external agents, although, as shown by A. V. Kibyakov (1961), section of the postganglionic sinus nerve of a frog causes a cessation of the automatic action of the heart after 5-9 days. This property of automatism is characteristic for the conductive system only; the contractive myocardium apparently lacks this property. The rhythmical processing of impulses in the heart of a mammal is connected with the function of the myocardium and with the function of the nerve structures within it. The neurogenic theory of the origin of cardiac impulses has now lost its strength, and the myogenic is already generally accepted. K. M. Bykov and his collaborators are

justified in asserting that the dispute between the neurogenic and myogenic theories of automatism is somewhat academic because within a heart, which is a single organ, there is a close interaction of all of its components.

The property of automatism is manifested to different degrees by the different components of the heart. From this point of view, it is possible to distinguish three levels of automatic centers. In the upper level, there is the automatic center of the first order - the sinusoidal node. Under normal conditions, this node can produce an average of 70-80 impulses per minute. In the middle level, we find the automatic center of the second order - the atrioventricular node, capable of producing an average of 40-50 impulses per minute. In the lower level, localized in the ventricles and including the His system, there are automatic centers of the third order with the capacity of producing an average of 10-20 impulses per minute. It can be seen that under normal conditions the function of automatism is strongest in the upper levels and becomes gradually weaker as we go down to lower levels.

Under physiological conditions, the cardiac rhythm is of sino-auricular origin. This is demonstrated by many experimental facts. Thus, during the time of embryonic development, the earliest heart activation is observed precisely in the region of the sinusoidal node. In the dying heart, this region is the last to stop functioning; in a normally contracting heart the first electrical potentials appear in the sinusoidal region. If, for various reasons, the function of the sinusoidal node is suppressed or disappears entirely, the automatic centers of the second order take over. With the disruption of these centers, those of the third order come into action. Thus, according to a very apt expression by Jouve, Sene and Pierron (1954), a heart has a hierarchy of automatic centers. /18

In addition to the described automatism of the heart, several authors hold the so-called "dualistic" concept, according to which there are two centers of automatism, independent of one another: one for the auricles and one for the ventricles of the heart. Certain authors assume a variant of this. They think that, although there are two active independent centers, the atrioventricular node needs a longer period of time to produce an impulse than needed by the sino-auricular node; therefore, the next sino-auricular impulse in the series discharges that of the atrioventricular node. V. S. Salmanovich (1954) finds that in the apex of the atrioventricular node, an interruption of the excitation occurs and is followed by the formation of a new stimulus. All these questions are still disputable. In our work, we adhere to the classical description and generally accepted concepts of cardiac automatism.

2. Excitability

Living tissue has the property of responding to various internal or external stimuli. All cardiac components have this property, but it is comparatively stronger in the nodal tissue. The function of cardiac excitability has certain characteristic peculiarities. When a stimulus of adequate intensity acts on the heart, the latter responds with a maximum contraction acting like a whole unit. An increase in the force of the stimulus does not

produce an increased response of the heart. In this respect, the myocardium differs sharply from a skeletal muscle, for which the force of contraction depends on the intensity of the applied stimulus. This rule of the heart, which is known in the literature under the name "all or none", depends on the syncytial structure of the myocardium. Therefore, any impulse of threshold intensity causes the contraction of all the muscular fibers, and a stimulus of a strength lower than this threshold intensity does not produce a contraction. This rule is not without certain flaws; for example, the force of contraction of the myocardium is not always the same, it depends on a series of physiological factors (see below).

The function of cardiac excitability is distinguished by another peculiarity. In contrast to skeletal muscles, the myocardium does not fall into a tetanic contraction and very often its response to impulses which rapidly follow each other is a single contraction. This phenomenon is explained by the law of the periodic refractivity of the myocardium. In muscular and nervous tissues, after each flare of stimulation, there is an absolute refractory period (a period of complete nonexcitability), followed by a phase of reduced excitability gradually returning to normal excitability. This is the relative refractory period. The absolute refractory period of the heart muscle is quite lengthy; therefore, the heart is incapable of producing a tetanic contraction. In a normally contracting heart, it coincides, or, as noted by L. I. Vogelsson (1957), almost coincides with the length of systole. Depending on the rhythm and the force of contraction, its length varies within the limits of 0.3-0.4 seconds in the ventricles and 0.06-0.12 seconds in the auricles. The slower the rhythm of cardiac pulsation, the longer the refractory period, and vice versa. The maximum length of the refractory period is observed in the atrioventricular node, the minimum - in the auricles. The ventricles are in an intermediate position in this respect. /19

The most important and practical part of the excitability function is the fact that the processes of appearance and propagation of impulses are accompanied by the development of electrical phenomena, which are taken as the basis for the electrophysiological investigations of cardiac activity.

3. Conductivity

This function pertains to both the contractile part of the myocardium and the nodal tissue. In the latter, it is more pronounced. Conductivity is a very stable property of the heart, and under normal conditions the impulse is transmitted from one point to the next without loss of intensity (Wright, 1957). The potentials registered at different points of the atrioventricular system show that the process of transmission is an electrical process and not a chemical one (Scher et al. 1954). The transmission of the impulse is at different speeds in the different sections of the conductive system. The maximum speed of 4000 mm/sec is observed at the level of the Purkinje cells or in the pseudo sinews of the conductive system, and the minimum of 200 mm/sec in the atrioventricular node. In the auricles the average speed is 300 mm/sec and in the subendocardial layers the speed is 3000-4000 mm/sec. Normally, an impulse passes through a distance of 15 cm in the auricle and 20 cm in the ventricle. Sano, Takayama and Shimamoto (1959) point out that the direction of the muscular fibers plays an important role in the speed of impulse /20

transmission. The transmission of an impulse is several times more rapid if it goes in a direction parallel to the direction of the muscular fibers and not in the direction vertical to their axes.

After its appearance in the sino-auricular node, the impulse propagates spherically and radially in both auricles and reaches the atrioventricular node. From here, after a short pause, it propagates through the His system into the ventricles. The process of gradual excitation of the ventricles is of great theoretical and practical interest and was quite well investigated in the work of Lewis (1925). The middle part of the interventricular partition is the first part to be involved in the process, because it is there that the impulse is transmitted through the left bundle branch of the bundle of His; from here, the wave of excitation proceeds almost perpendicularly to the partition and forms an initial vector of the electrical forces in the left-to-right direction. After this, it propagates into the right ventricle. Now the walls of the ventricles become excited--first the papillary muscles and then the lateral walls. At all levels of the ventricles, the impulse passes from the endocardial surface of the myocardium to its epicardial surface. Therefore, the subepicardial layers are excited somewhat later than the subendocardial layers. This phenomenon is explained by the anatomical arrangement of the network of Purkinje cells; the peculiarity of the excitation impulse propagation is due to this factor (Medrano et al. 1960). In general, it may be stated that in the ventricles the impulse propagates upward. Therefore, the apex of the heart is involved in the excitation process somewhat earlier (by 1/100 sec) than its base.

Moreover, the transmission of an impulse always goes by the same paths. Pibberger (1957) demonstrated that in contrast to the impulse propagation through the subepicardial layers, the order of impulse propagation in the subendocardial layers is irregular.

4. Contractility

While the force of contraction in a skeletal muscle depends on the strength of excitation (up to a definite magnitude), this is not true for cardiac muscle. In this case, Starling's "law of the heart" holds. According to this law, the force of contraction of the heart muscle is directly proportional to the initial (prior to contraction) length of the muscular fibers. This proportionality indicates the high development of the quality of biological adaptability possessed by the heart. This is so because the initial length or extension of the heart fiber in the diastolic phase depends on the supply of blood into the heart chambers. The greater the supply, the stronger the contraction. Naturally, other factors in the circulation such as, for 21 example, the blood pressure in the pulmonary and systemic circulation, the duration of diastole in the preceding cycle, the conditions of blood supply to, and the general state of nourishment of the myocardium affect the magnitude of cardiac contraction. A definite significance is also attached to the strength of the nerves of the heart (I. P. Pavlov) which, while affecting the trophic state of the myocardium, regulate the metabolic processes and improve the contractile energy. (The details of contractility are found in the section on "Mechanical Activity of the Heart".)

Should we consider the tone of the heart as a separate (fifth) function? In our opinion, this should not be given special consideration because, after all, the tone is only a manifestation of the function of contractility in the diastolic phase. Unfortunately, the known methods do not provide the possibility of an objective tonic study under clinical conditions. L. I. Vogel'son (1957) finds that the tone function characteristic of a vascular system must also act within the heart, because both phylogenetically and ontogenetically, the heart and the vascular system represent a single whole unit.

IV. CARDIAC INNERVATION

The heart is innervated by fibers of the sympathetic and parasympathetic sections of the autonomic nervous system.

The cardiac fibers of the vagus emerge from the medulla oblongata and arrange themselves within the stem of this nerve prior to its entrance into the chest region. At this level, the nerve is divided into the upper, middle and lower cardiac nerves, with their preganglionic fibers ending in the cellular colonies of the myocardium. The right vagus serves basically for the innervation of the sinusoidal node, while the left vagus terminates for the most part within the atrioventricular node. The fibers serving for the innervation of the auricles, the bundle of His and the base of the heart are generated from the nerve cells found in these two nodes (Wright, 1947).

The preganglionic sympathetic fibers emerge from the spinal cord within the three upper, anterior thoracic roots, after which they enter the sympathetic trunk and terminate in all three cervical ganglia, for the most part in the cells of the upper thoracic sympathetic stellate ganglion. The post-ganglionic fibers begin at these points and reach the heart as the cardiac branches of the sympathetic nerve.

The strongest effect of the parasympathetic and sympathetic nerves is produced on the auricles. The apex of the heart does not receive any of the vagus fibers. However, as was pointed out by Raab (1959), the opinion that the ventricles of the heart are totally devoid of elements of the vagus and are therefore free of the direct retarding action of the vagus, cannot be definitely accepted. V. A. Shidlovskiy (1960) reports that, although basically the work of the ventricles is regulated by the hemodynamic interaction between the auricles and the ventricles, a direct effect of the extracardial nerves cannot be excluded in the case of very strong stimuli.

In addition to the efferent neurons, there are also efferent heart fibers of various types. According to Sheffer (B. S. Kulayev, 1961), in the composition of the extracardial nerves there are 5-6 times more afferent 22 fibers than efferent ones. Among the afferent fibers, there are also sensory fibers transmitting pain impulses from the receptors of the myocardium through sympathetic cardiac nerves to the cervical ganglia or to the upper thoracic ganglion (Raab, 1959).

After E. Weber and G. Weber (1845) described the retarding action of the vagus, and I. Zion and M. Zion (1866) demonstrated acceleration of the heart rhythm after stimulation of the sympathicus, the question of the influence of these nerves on the activity of the heart became the subject of comprehensive and detailed study. In classical physiology, it is assumed that both vagus and sympathicus produce an antagonistic effect on the work of the heart. It is thought that the vagus produces, in general, a negative chronotropic, bathmotropic, dromotropic and inotropic action, while the sympathicus acts in the opposite sense. This question, however, is not yet finally resolved. Thus, L. I. Vogel'son (1951) notes that the character of the action of these nerves changes depending on the direction of impulses (from the right or from the left nerves). Bouckaert and Heymans (1937) demonstrated that the function of the vagus consists not only of a retardation effect, but that its excitation may also accelerate the heart rhythm.

N. L. Yastrebtsova and M. G. Udel'nov (1955) report that under the influence of the reflex action of the vagus, the level of polarization of the myocardium is altered and that the vagus may both accelerate and decelerate the heart rhythm. They arrive at the conclusion that the accelerating reflexes on the heart are caused simultaneously by both sections of the autonomic nervous system, although the effect of these reflexes depends on the various conditions created within the organism. The sympathetic innervation becomes involved in the case of the generalized reflexes, while the parasympathetic innervation produces a specific reflex action. The study of the stellate ganglion in dogs made by Herrmann, Jourdan, and Froment (1938) demonstrate that the sympathetic innervation may produce a decelerating effect on the heart rhythm. But Knox and McDowall (1956), with reference to observations made by Claude Bernard, assert that an adequate stimulation of almost every nerve will produce a deceleration of the rhythm.

In the regulation of the heart rhythm, a definite role is also played by the reticular formation of the brain-stem (F. P. Vedyayev and V. I. Klimova-Cherkasova, (1961); A. A. Volokhov (1961).) Furthermore, many internal (Binebridge reflex) and external cardiac reflexes (such as those from the baro- and chemoreceptors of the aorta and carotid), the Gering and Zion-Ludwig nerves, the pulmonary circulation, etc., are of great significance (Salisbury et al., 1959, etc.). In this respect, the experiments of A. V. Trubetzkoy (1961) and those of Kulayev (1961) are of great interest, where it was shown that the heart itself is a rich compensating buffer and reflexogenic zone for the whole cardiovascular system. We agree with the opinion of M. G. Udel'nov (1961) that the organization of the finite neuro-action depends not only on the nervous system, but also on the structural and functional peculiarities of the nervous periphery and on the organ itself.

V. VASCULAR SYSTEM OF THE HEART

The coronary circulation of the heart is characterized by a series of anatomical and physiological peculiarities. This prompts certain authors to regard it as the third distinct path of blood circulation (B. V. Ognev, V. N. Savvin and L. A. Savel'yeva, 1954; A. M. Sigal, 1955). Such an

opinion is unacceptable, however, because, as was pointed out by V. V. Parin and F. Z. Meyerson (1960), these peculiarities alone cannot serve as a criterion for this assumption, considering the fact that the blood supply system of almost every organ has some peculiarities of its own.

Beginning from the right aortic sinus, the right coronary artery first proceeds forward and to the right, between the root of the pulmonary artery and the right auricle, then downward to the point of juncture of the right and lower heart edges; it continues at this level into the rear surface of the heart forming the ramus descendens posterior. The left coronary artery starts from the left aortic sinus, goes somewhat forward arranging itself between the pulmonary artery and the left auricle, then to the left toward the left part of the coronary sulcus. On this level it separates itself into two branches - the ramus descendens anterior, proceeding along the anterior longitudinal sulcus toward the apex of the heart, and the ramus circumflexus, passing into the rear surface of the heart. Although considerable variations are observable in the normal vascularization (three types are observed) of the right and left ventricles, it may be stated generally that the right coronary artery feeds the right ventricle and the larger part of the transmission system, while the left coronary artery feeds the left ventricle. /23

The coronary sinus and anterior coronary veins form the most important paths of the venous circulation of the heart. The coronary sinus is located in the rear part of the coronary sulcus between the left auricle and left ventricle. It has a length of about 2-3 cm and terminates in the semi-lunar valve in the right auricle between the point of entrance of the lower vena cava and the atrioventricular opening. According to the experimental results of Gregg and Shipley (1947), the principal drainage system of the left ventricle is the coronary sinus. The right ventricle is drained by the anterior heart veins. Between the two systems there is an extensive network of anastomoses.

The characteristic anatomical peculiarity of the coronary circulation resides in the fact that it encircles the heart chambers without being a closed system. It is quite often interrupted by collateral vessels, both in the arterial part of the capillary system (arteriololuminal and arterio-sinusoidal vessels) and in its venous part.

In the literature, there are quite different opinions with regard to the finiteness of the coronary vessels. Anatomical investigations demonstrate the presence of anastomoses between small branches and between the capillaries of two coronary arteries. Nevertheless, in spite of the existence of many intramural anastomoses of several orders, in the presence of severe disruptions, the veins can be clinically considered finite (B. B. Ognev, V. A. Savvin and L. A. Savel'yeva, 1954).

Alella (1956) notes correctly that the coronary blood flow can be considered a result of the interaction of mechanical, metabolic, nervous and humoral factors. According to Gregg (1950) the intramural blood flow is controlled by two principal factors - the condition of the vascular con-

traction and their external non-vascular support. V. V. Parin and F. Z. Meyerson (1960) point out the following circumstances which determine the "privileged conditions" of blood supply to the myocardium (on the average, this supply is 10 times stronger than that of other organs): the level of metabolic processes in the myocardium, tone within the coronary arteries, aortic pressure, heart rhythm and pressure within the heart chambers. It should be noted that the coronary influx occurs during the diastolic phase and the efflux during the systolic phase of the ventricles. The nervous regulation of the coronary circulation is of great importance. In this connection, many known facts do not correspond to the general physiological assumption that stimulation of the vagus causes vasoconstriction and, consequently, a decrease in the coronary circulation, and that stimulation of the sympathicus produces an opposite effect. Thus, Katz and Jochim (1939) assert, on the basis of original experiments (with the isolated head and fibrillating heart of an animal), that sympathetic fibers produce both contracting and dilating effects on the vessels. They add, however, that the prevailing effect of adrenergic innervation is that of contraction of the vessels. Scott's and Balourdas' experiments (1959) on dogs demonstrated that after vagotomy and administration of atropine, there occurs a considerable increase in coronary blood flow and an acceleration of heart rhythm accompanied by a decline in the efficiency of the left ventricle. Green and Kepchar (1959) find that the coronary blood flow is determined by the contraction of the ventricles and the tone of the coronary arteries. The coronary blood flow increases under the influence of the sympathicus and under that of many adrenergic substances, and is not under cholinergic control. The tone of the coronary arteries is not subject to either sympathetic or cholinergic control. Gregg and Shipley (1944) explain the increase in coronary blood flow occurring in conjunction with an overloaded left ventricle by the appearance of a large quantity of local metabolites and/or by a relative anoxia as the result of an increased expenditure of oxygen. Thus, the question of nervous regulation of the coronary circulation is still in need of further investigation. One may agree with Folkow (1956) who asserts that it is very difficult to determine to what extent various alterations in the coronary circulation depend on the influence of a specific vessel-moving fiber or on an indirect action of vasodilating metabolites. It must be kept in mind in this connection that quite a significant role is played by certain humoral agents (acetylcholine, adrenalin, noradrenalin, etc.) and, as noted by A. V. Tonkikh (1961), by neurohumoral chain reactions. /24

VI. ELECTRICAL ACTIVITY OF THE HEART

In biology, electrical recording may be used to measure the potentials generated in the biological elements and the resistance to distribution of the externally applied current within and outside of the tissue cells (Curtis, 1956). In electrocardiology, we deal principally with the measurements of the first group.

In biology, considerable consideration has been given to electrical phenomena taking place in various organs and tissues since Galvani's observation in 1786 that, when closing an electric circuit between the nerve and

leg muscle by means of a metallic conductor, a muscular contraction occurs. Galvani pioneered the concept of the generation of electricity in living tissue, while his contemporary Volta believed that the electric currents are generated as a result of the passage of metallic conductors through the saline media.

This argument became a foundation of a new chapter in physiology - electrophysiology. Its initial period is connected with such names as Duboi-Raymond, I. M. Sechenov, N. Ye. Vvedenskiy, Einthoven, etc. Two cornerstones in the development of physiology were: introduction of the cathode ray oscilloscope by Eilanger and Gasser in 1922; application of the micro-electrode technique to the investigation and study of transmembrane potentials. This in turn created the possibility of electrophysiological research at the cellular level (Bures, Petran and Zachar, 1960).

1. Basic Principles of Electrophysiology

The basic electrophysiological processes occurring within the heart consist of excitation of muscle cells and transmission of impulses. As was shown by Hoffman and Kreinfield (1962), certain specialized membranes possess the property of excitation. These membranes are also characterized by the ability to transmit ions. This transmission depends on the fact that, in addition to excitation-prone membranes, the cells possess an electrical ability to transmit the impulses to adjacent regions. In the process of impulse transmission, considerable significance must be assigned to the electrical properties of the extracellular fluid, cytoplasm and membrane. /25

Even in 1848, Duboi-Raymond noticed that, when two electrodes are placed on the sural muscle of a frog, both electrodes register the same potential. However, after damaging a certain area of the muscle, a potential difference appears between the intact and damaged surfaces. The potential of the intact surface was found to be electronegative. This phenomenon was named the "resting potential" or "current of injury."

The process of excitation in nerves and muscles is accompanied by the appearance of electrical phenomena; the area excited at a certain moment becomes electronegative with respect to the area not yet excited. In this manner, a potential difference and the generation of electromotive force appears. This phenomenon is known under the name of the "action potential." Registration of the action potential serves as an electrical indicator of the passage of the impulse through a muscle or nerve. In the heart muscle, the action potential has the peculiarity of beginning prior to contraction and continuing to the end of contraction with very little loss of its initial intensity (D. S. Vorontsov, 1961); the comparatively long duration of this current ensures a prolonged contraction of the heart muscles.

The resting potential in the majority of cardiac fibers is about 85-95 mv, while the action potential is about 100-120 mv (Hoffman and Kreinfield, 1962).

Electrophysiological experiments show that, after reaching a peak, the

action potential curve approaches the isoelectric line with a decelerated speed. This lingering positive potential is called the "after potential."

An individual muscle fiber responds with maximum force to a stimulus of threshold intensity. This is the so-called "all or none" law which, according to K. M. Bykov and his co-workers, does not correspond to the real state of affairs. These authors argue that gradation of muscular contractions within the limits of a motor unit (an individual nerve fiber with a group of muscle fibers innervated by it) is attained mostly by an increase in the frequency of stimuli; in addition, the gradation of contraction of the whole muscle also depends on different numbers of acting motor units.

In the development of electrophysiology, the works by N. Ye. Vvedenskiy /26 and later those of A. A. Ukhtomski were of great significance. They defined the concept of lability of a tissue as the maximum number of impulses which a given tissue can reproduce per second in a definite correspondence with the rhythm of applied stimuli. In connection with this, the phenomenon of parabiosis with the equalizing, paradoxical and retarding stages has been described. The phenomenon of exaltation described by N. Ye. Vvedenskiy coincides with the phase of increased excitability which appears after a relative refractory phase.

2. Generation Mechanism of Resting and Action Currents

I. M. Sechenov attached considerable significance to metabolic processes in the generation of the electromotive force in tissues. On the basis of this principle and applying the Arrhenius theory of electrolytic dissociation, V. Yu. Chagovets assumed that with the activation of these processes there occurs an increase in the quantity of carbon dioxide, whose dissociation into ions causes the difference in potential in a tissue.

In this connection, the so-called membrane theory acquired considerable importance. Numerous experimental observations of isolated vegetable and animal cells show that, when the cell is at rest, there is a potential difference between the outer and inner surface of the membrane, so that inside the cell there is a negative electrical charge with respect to the outside surface. Such a cell is known as a polarized cell (Fig. 2a).

Berstein (1902) reported that in a state of rest the cellular membrane is permeable to cations (such as potassium) and not to anions (such as chlorine). Thus, two layers of ions are formed which interact electrostatically: a layer with positive ions forms on the outer surface of the membrane and a layer with negative ions forms on the inner surface so that, according to Curtis and Cole (1941), in a state of rest the average membrane potential is about 51 mv. There is a potential difference between these two layers, but there is no electromotive force because the layers are separated by the impenetrable membrane acting as an insulator. According to the latest information, the resting potential is a result of the tendency of potassium ions to diffuse from the region of high concentration within the muscle fibers into the region of lower concentration on the outer surface of the fiber (Hoffman and Kreinfield, 1962).

According to the membrane theory, the resting potential is produced as a result of diffusion of negative ions from within the zones of injured tissue outward. Such a zone becomes electronegative with respect to a normally polarized area. The action potential is produced as a result of the fact that a stimulus produces an increase in the permeability of the membrane and its resistance is reduced. Thus, its conductivity is increased and both anions and cations diffuse through the membrane. In such cases, the potential on the outer surface of the membrane may become as large as 110 mv (Curtis and Cole, 1941). Thus, the area which is excited, at a certain moment, becomes electronegative with respect to the remaining normally polarized surface, which in turn produces the electromotive force. With the cessation of excitation, the cell returns to its initial polarized state. Weidmann (1961) found that excitation is connected with introduction of Na^+ ions, while recovery is associated with escape of K^+ ions. It is interesting that Van der Werff (1948) explains the processes connected with the motion of ions by phenomena of assimilation and dissimulation. In fact, he bases his conclusions on the opinion of I. M. Sechenov.

Thus, the process of gradual envelopment of a cell by excitation can be regarded as a depolarization, and that of its return to the state of rest as repolarization.

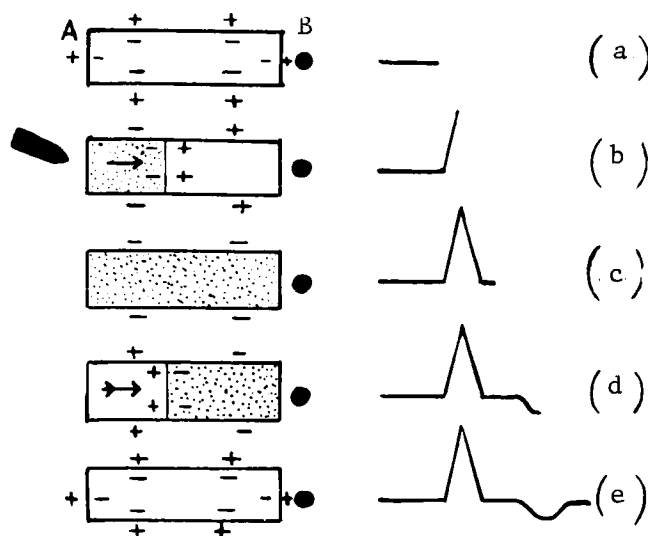


Fig. 2: Process of Excitation of a Muscle Cell*: (a) cell in a state of rest - polarized state; (b) beginning of depolarization phase; (c) complete depolarization of cell - stress phase; (d) beginning of repolarization phase; (e) cell after full repolarization - polarized state.

*In all sketches, the muscle cell (or muscular fiber) is represented by a rectangle; dotted surfaces indicate the excited part of the cell; the heavy arrow indicates the location of excitation; the black circle represents the probing electrode of unipolar recording; the thin arrow represents the direction of the process of depolarization; the thin arrow with a tail represents the direction of the process of repolarization.

In the heart, the duration of these phenomena, especially that of repolarization, is considerably longer than in other excitable tissues. These processes can be represented graphically (Fig. 2). A muscle fiber is attached to a galvanometer by electrodes and conductors. The galvanometer is supplied with a measuring device. Area A is subjected to excitation and a process of depolarization begins which propagates gradually from point A toward point B. During this period, the galvanometer registers an electric wave which rises from the isoelectric line, reaches a peak and descends to this line. When a cell returns to the resting state, a current in the opposite direction is registered, and the curve shows a wave in the opposite direction - this is the phase of repolarization. Thus, a biphasic curve appears with waves in opposite directions divided from one another by a small isoelectric interval. This interval coincides with the maximum voltage within the cell, when the muscle is in a charged state after a complete depolarization. But since there is no potential difference, no electromotive force is produced; this is followed by the phase of repolarization. /28

In electrophysiology, there is also a monophasic curve (Figs. 3, 48) which is registered when one electrode is placed directly on the outer surface of the cell and the other inside the cell or on a point on its surface which cannot be reached by excitation (this can be achieved by means of mechanical, chemical or thermal injury to the tissue or by means of local cooling of the cell to 0°C).

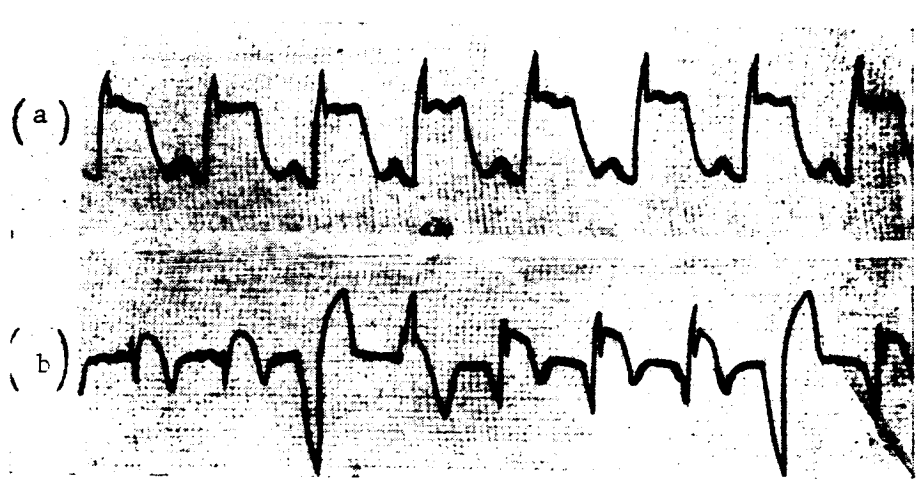


Fig. 3: Monophasic curve: (a) curve obtained after artificial damage to the rear surface of the left ventricle in the heart of a dog; (b) curve obtained one day after ligating the coronary artery of an experimental dog. (In all electrocardiograms the time registration period is 0.02 sec.)

The monophasic curve reflects the excitation of only the undamaged part of the cell; a sharp upward oscillation corresponds to depolarization of this part. The plateau corresponds to the phase of complete depolarization or, as we say, to the tension phase; the slow descent indicates the phase of /29

gradual repolarization (Lenegre, Carouso and Chevalier, 1954). Under clinical conditions, the monophasic curve generally appears in myocardial infarction; quite often we also encounter this curve during heart surgeries and in experimental work manifesting damage to the myocardium (Fig. 3).

3. Some Practical Problems in Cardiac Physiology

The phenomena described above were observed during experiments with an isolated muscle cell or tissue. There is great interest for a clinician in experiments conducted under conditions approaching a normal state, i.e. when the tissue which is placed within a volume conductor is homogeneous and sufficiently stretched. In such a case, electrodes can be placed into the medium of the conductor and not directly on the muscle surface. With an increase in the distance of the electrodes from the muscle tissue, the number of potentials recorded decreases.

All concepts with regard to the electrical properties of tissues arranged in a volume conductor are based on the dipole theory, the basic principles of which were formulated by Craib (1928). According to this theory, propagation of excitation and restoration within a muscle bundle situated within a volume conductor can be regarded as the motion of a dipole along its surface, whereupon during depolarization the positive pole of the dipole is directed forward and the negative backward. During repolarization the directions are reversed: the negative pole is directed forward and the positive backward (Fig. 4a, b). In the volume conductor, the isopotential lines are on the side of the positive pole of the dipole and the negative lines on the side of its negative pole (Fig. 4a). These lines are separated from one another by a perpendicular line passing through the point of junction of the two poles, where the potential is equal to zero.

Although the dipole theory is generally accepted, it may still be criticized. There are other interpretations, but we shall retain the dipole theory because it provides an explanation of many phenomena pertaining to the electrical activity of the heart. In addition, it is of definite practical significance, which we shall endeavor to demonstrate further on in this work.

As was noted by Sodi-Pallares (1956), the method of investigation of electrical phenomena within a muscle bundle situated inside a volume conductor allows a wide range of selection of areas for application of the electrodes. Thus we can distinguish direct testing, when the electrodes are placed directly on the surface of the muscle bundle, semi-direct testing, when the electrodes are placed at a relatively small distance from the bundle but not in immediate contact with it, or distant testing, when the electrodes are placed at longer distances from the bundle. All of these leads may be bipolar or unipolar. /30

In the case of bipolar leads (Figure 5a), two electrodes are placed directly on the muscle surface or within a volume conductor at equal distances from the muscle. In this case, both electrodes are active (ex-

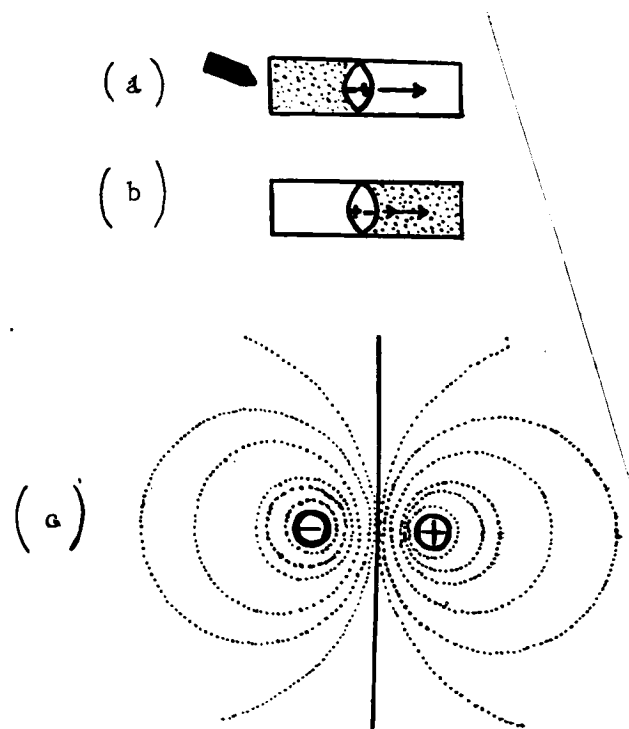


Fig. 4: The Process of Excitation of a Muscle Bundle According to the Dipole Theory: (a) phase of depolarization; (b) phase of repolarization; (c) isopotential lines about a dipole.

ploring) and each registers the potential at the point of its location. In unipolar testing (Fig. 5b), one electrode is placed very close to the muscle bundle and the second at a great distance from it. Then, for all practical purposes, the first electrode is active and the second is neutral and the registered curve is almost entirely a reflection of the potential in the area of the position of the active electrode. It must be noted, however, that /31 theoretically a lead of this type is not exactly unipolar because the second electrode, no matter how distant, may still register some of the potential. There exists another, more correct, principle for obtaining the unipolar lead (Fig. 5c). In this case, three electrodes are placed on the periphery of the volume conductor so that their distances from each other and from the bundle are equal. When these electrodes are connected by conductors, a so-called central terminal conductor appears whose potential is zero (for details see page 50). Thus, a neutral electrode is obtained. Then, by placing an active electrode at a required point, it is possible to register the resulting unipolar lead.

It has been stated that the curve of the action current consists of two equal but directionally opposed phases. Using the dipole theory and the unipolar method of recording, it is possible to demonstrate that the configuration of the curve depends on the series of conditions whose changes determine the displacements in the shape of the obtained curve. These questions were thoroughly investigated by Goldberger (1954), and we have made use of the results of his investigations.

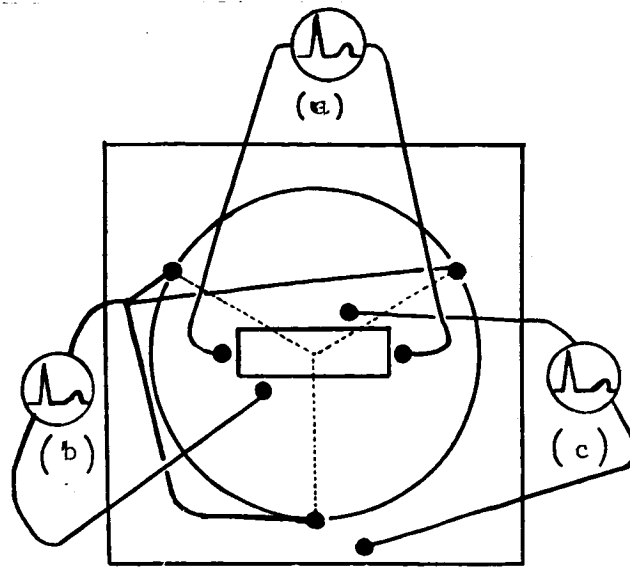


Fig. 5: Bipolar and Unipolar Leads: (a) bipolar lead; (b) unipolar lead where the second electrode is placed at a large distance from the muscle bundle; (c) unipolar electrode with the application of a central terminal conductor.

Above all, it is necessary to recognize the following general conditions: the exploring electrode of a unipolar lead records positive oscillations in a direction going upward from the isoelectric line when it is facing the normal (unexcited) muscular surface, and negative oscillations in a downward direction when it faces the excited surface of the muscle. Knowing this, the following phenomena may be discussed: /32

The effect of the direction of stimulus propagation on a unipolar lead. In Figs. 6a, b, c, it can be seen that a curve with reversed phases is obtained when the direction of stimulus propagation is changed.

The effect of the direction of repolarization of the excited muscle on a unipolar lead. Figs. 6a, b, c, d, demonstrate that when a muscle returns to a state of rest from the same side as that of the start of stimulus propagation, the curve phases are opposite to each other; but when this process starts from the opposite side, the registered curve is composed of two waves having the same direction.

Effect of the position of the exploring electrode on uniform leads. This is illustrated in Fig. 6f. The configuration of the curve changes depending on the position of the electrode. It must be taken into consideration that the distance of the electrode from the muscle bundle is also of significance, but only with regard to a decrease in amplitude with increased distance of the electrode. /33

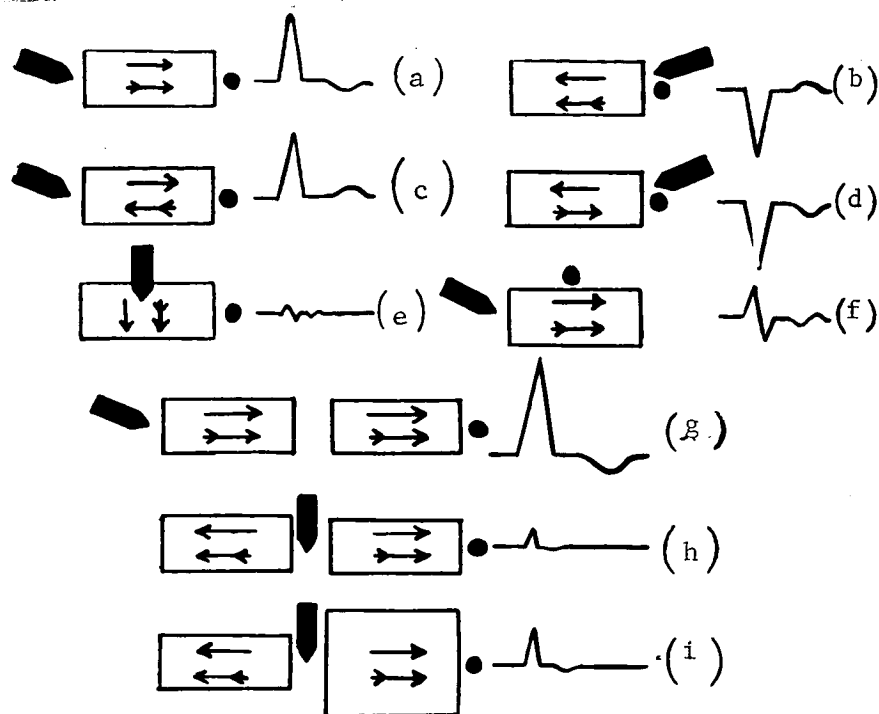


Fig. 6: The effect produced by various factors of the excitation processes within a muscular bundle on the shape of the obtained curve. (Explanations are given in the text.)

Effect of simultaneous stimulus propagation along certain muscular bundles. Fig. 6g, h, i show that, if the stimulus propagates along two muscular bundles (with equal volumes) in the same direction, the amplitude of the obtained curve is almost doubled; in the other case, the curves in opposite directions obtained from separate muscles neutralize each other, or a small oscillation appears corresponding to the potential of the bundle which is closer to the electrode; the mass of the bundle also has an effect

VII. MECHANICAL ACTIVITY OF THE HEART

The first experimental data with regard to the mechanical activity of the heart were obtained at the time when physiologists first recorded the diastole and systole of an animal's isolated heart. However, such a simplified concept of cardiac contraction could not be of much value, especially in the development of clinical investigations. For a certain period of time, the rapid development and wide application of methods for the study of the electrical phenomena of cardiac activity neglected this most important aspect of the heart mechanism. During the last 20-30 years, however, questions pertaining to the mechanical aspects of cardiac contraction began to be intensively investigated by physiologists and clinicians. At present, they are recognized as having an important significance in modern cardiology and, as such, occupy the central place in scientific investiga-

tions.

During the contractile phase of the heart, certain mechanical oscillations appear, two types of which are of great importance from the standpoint of synthetic electrocardiology. The first type comprises the oscillation of the chest wall. These can be of either low frequency (10-15 per second) or relatively high frequency (600-800 and 1000 oscillations per sec.). The low-frequency oscillations include those from the apex thrust, while the high-frequency oscillations include those due to the sonic phenomena of the heart. The second type consists of ballistic oscillations with various parameters of motion propagating along all axes of the body. These mechanical oscillations are minutely discussed in the corresponding chapters of this book (see the chapters on "Phonocardiography", and "Ballistography").

The phases of cardiac contraction can be successfully studied by measuring the blood pressure in the heart chambers and by the cardiometric method of determining the changes in the volume of the heart. In order to record curves pertaining to blood pressure within the inner chambers of the heart, manometers of special construction are used. These experimental problems were well explored in investigations by Wiggers, (1952). Investigation of the blood pressure within the heart chambers under clinical conditions became possible after the introduction of heart catheterization (Forssmann, 1929; Cournand, 1942-1945, 1949, 1957; Richards, 1957; Ye. M. Meshalkin, 1954; V. S. Savel'yev, 1961, etc.). We shall give a description of the phases of mechanical activity of the heart in the chronological order of one cardiac cycle (Fig. 7).

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1. Auricular Systole

The normal cycle of the heart begins with the contraction of the auricles. The contraction wave begins at the level of the openings of the vena cava and progresses downward. During this period, there is no reflux of blood from the auricles to the veins because of a timely contraction of muscles surrounding the site of entry of these veins into the auricles and a considerable amount of pressure within them.

The pressure in the auricles begins to increase, but reaches only a value of 2-5 mm Hg. This is due to the fact that the atrioventricular valves are then open for the passage of blood into the ventricles. Auricular systole lasts for about 0.1 sec. and is followed by auricular diastole, which lasts until the start of auricular systole in the next cardiac cycle.

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2. Ventricular Systole

After the end of auricular systole, ventricular systole begins. During this systole, there is a decrease in all diameters of the heart, and an expansion of the large vessels as a result of a downward pull of the base of the heart. The left ventricle begins to contract somewhat later than the right ventricle; for all practical purposes, however, it may be assumed that their contraction is synchronous. Ventricular systole consists of two phases: a phase of isometric contraction (or tension) and the phase of

blood expulsion.

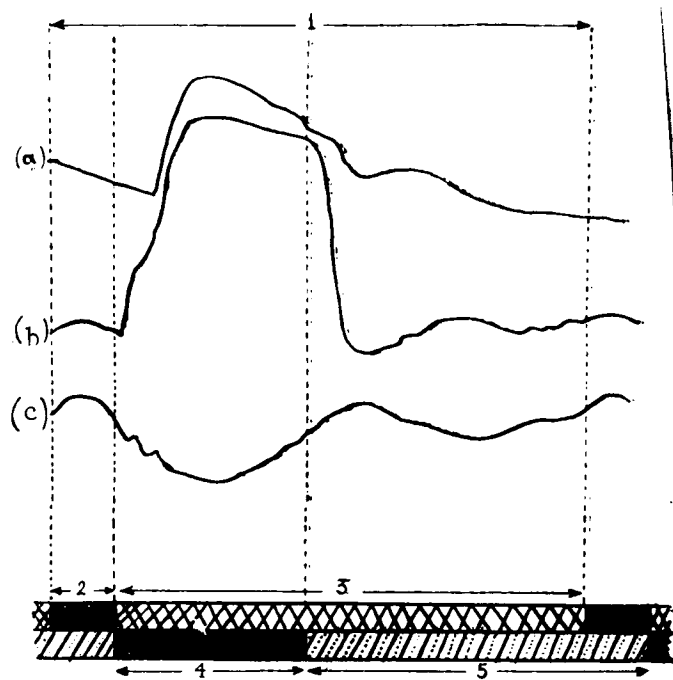


Fig. 7: Schematic representation of the phases of cardiac contraction: (a) sphygmogram of the carotid; (b) curve of blood pressure within the left ventricle; (c) curve of blood pressure within the left auricle; (1) one cardiac cycle; (2) auricular systole; (3) auricular diastole; (4) ventricular systole; (5) ventricular diastole.

A. The tensile phase or phase of isometric contraction. Prior to the advent of ventricular systole, the pressure within the auricles is almost equal to that within the ventricles, and the atrioventricular valves hang loosely over the surface of the blood within the ventricles. With the advent of systole the pressure within the ventricles begins to rise. At first this increase proceeds slowly, but later, with the contraction of all the fibers of the ventricular muscle, the increase becomes very rapid. When the pressure within the ventricles rises to a value of 2-6 mm Hg, the atrioventricular valves close and enter the auricles as dome-like protuberances. This increases the pressure within the auricles. Further motion of these valves into the auricles is hindered by the contraction of the capillary muscles, which contributes to the increase in pressure within the ventricles. Somewhat later, when the pressure within both ventricles becomes larger than in the aorta (prior to the advent of systole the pressure in the aorta is equal to 50-80 mm Hg), the semilunar valves within the pulmonary artery open, after which the phase of blood expulsion begins. Thus, the energy of isometric contraction of the ventricles is spent on the transformation of electrical into mechanical phenomena, the attainment of a high pressure within the ventricles, a slight deformation of the valves and a change in the shape and position of the heart. During this period, there

is no blood flow. Therefore, the tensile phase can be divided into two parts: the period of transformation and that of increase in pressure (see page 328). The duration of the tensile phase depends on a series of factors, such as the duration of the preceding cardiac cycle, the frequency of cardiac contractions, the stroke volume, peripheral resistance, etc. The average duration of this phase is from 0.05 to 0.10 sec.

B. The phase of blood expulsion. Wiggers reports that a contraction of the "base-apex" axis occurs during the tensile phase due to decrease in the length of the fibers in the partitions. This creates a sufficient vis a tergo to produce a vigorous and sharp expulsion of blood into the aorta. This is also aided by the gradual descent of the atrioventricular ring due to contraction of the papillary muscles and fibers in the interventricular partition. From the right ventricle, blood is expelled because of: contraction of the free wall of the ventricle, pressure of blood toward the interventricular partition, and lowering of the ring of the tricuspid valve. An important role is also played by the contraction of the interventricular partition. /36

From the left ventricle, blood is expelled as a result of decrease in the diameter of the ventricular cavity and shortening of the heart's axis. Expulsion of blood from this ventricle starts somewhat later and ends relatively sooner than in the right ventricle (Reshmer, 1961). Ventricular contraction is isotonic in character, which is manifested by a plateau-like curve of blood pressure. Blood expulsion is of short duration; this depends on a series of factors and is equal, on the average, to 0.20-0.25 sec. Cardiometric measurements show that the major part of the stroke volume (about 2/3) is expelled during the initial phase of blood expulsion; the rest - a small part - is expelled during the second period of this phase. Thus, the phase of blood expulsion may be regarded as composed of two periods:

a. The period of maximum or rapid blood expulsion. This initial period is characterized by a rapid outflow of blood from the ventricles into the corresponding arteries because of a sharp contraction of the ventricles. In the course of 0.05 sec., the internal ventricular pressure is raised to a maximum, reaching the value of 150 mm Hg. During this period, the dynamics of pressure in the aorta follow that of the internal ventricular pressure, but its level is somewhat lower than in the ventricle; the ventricle and aortic opening form an almost common chamber.

b. The period of reduced blood expulsion. This second period is characterized by the fact that certain portions of the ventricles cease to contract. Therefore, the pressure within the ventricle, and consequently that in the aorta, becomes lower. The outflow of blood through the branches of the aorta exceeds the blood supply to the aorta from the ventricle. The blood expulsion continues, however, because the pressure within the left ventricle is still greater than that in the aorta.

It must be noted that a small amount of blood remains in the ventricles after blood expulsion, the so-called residual blood (Luisada and Liu, 1956).

During the entire phase of blood expulsion, due to the gradual descent of the base of the heart - and that of the atrioventricular ring, the volume of the auricles is increased and the internal pressure in the auricles becomes lower. This creates favorable conditions for the suction of blood from the veins into the auricles. Under these conditions, the auricles are rapidly filled by blood since they are then isolated from the ventricles by closed atrioventricular valves.

3. Ventricular Diastole

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Ventricular diastole begins with a short period of protodiastole, when, after the end of ventricular systole the internal pressure in the ventricles drops rapidly to such an extent that the relatively high pressure in the aorta and in the pulmonary artery closes the semilunar valves. Also, as a result of the further relaxation of the muscles, the sharp pressure drop within the ventricles continues; the ventricles then become closed empty chambers, since both the semilunar and the atrioventricular valves are closed. This period is known as the period of isometric relaxation. When the pressure within the ventricles becomes less than that within the auricles, the atrioventricular valves open and the period of blood influx into the ventricles begins anew. It must be noted that the period of isometric relaxation is of differing duration in the right and left ventricles, because the tricuspid valve opens 0.05 sec. after the closing of the valve of the pulmonary artery, while the bicuspid valve opens 0.06-0.10 sec. after the closing of the aortic valve. Furthermore, Wigger (1945) distinguishes the phases of rapid filling, diastasis, and auricular contraction. Luisada (1953) notes that the period of blood supply to the ventricles during diastole passes through the following three stages:

Rapid, passive filling (early diastole) - this takes place early in diastole due to the pressure difference between the full auricles and empty ventricles;

Slow filling (diastasis) - this takes place in the middle of diastole due to resistance of the already partially filled ventricles to additional influx of blood from the auricles.

Rapid active filling (presystole) - this is due to the contraction of the auricles. This contraction can expel into the ventricles from 35 to 60% of the total mass of blood in the ventricles, depending on the duration of diastole. The shorter the ventricular diastole the larger this volume of blood, and vice-versa. The long period of diastole ends with the contraction of the auricles.

The picture of the dynamics of systole and diastole of the heart given above is schematic in character. We did not stop to discuss such topics as the significance of the condition of the coronary vessels, residual blood volume, the laws of cardiac activity, etc., which are discussed in detail by Wiggers (1952). These topics are of very great theoretical and practical interest and can be treated only in the specialized literature.

CHAPTER II

METHODOLOGY OF SYNTHETIC ELECTROCARDIOLOGY

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Based on the information in the previous chapter, it may be stated that the study of the electrical and mechanical manifestations of heart contraction makes accessible the delicate mechanism of heart action and a clear picture of the operation of the normal or the pathological heart. To the physiologist such a task does not present great difficulties. The various methods of clinical research study (for the most part) present only one or another aspect of heart activity. The full presentation of the phenomena described can give such clinical* guidance only when appropriate methods are applied simultaneously and concurrently and the data examined in accordance with a previously worked out plan. A specialist must have such a methodology because, as we noted in the introduction of the book, only then can a good and purposeful synthesis of the analytical material occur. We are convinced that such an approach, which constitutes the basis of synthetic electrocardiology, has broad possibilities for scientific and practical cardiology.

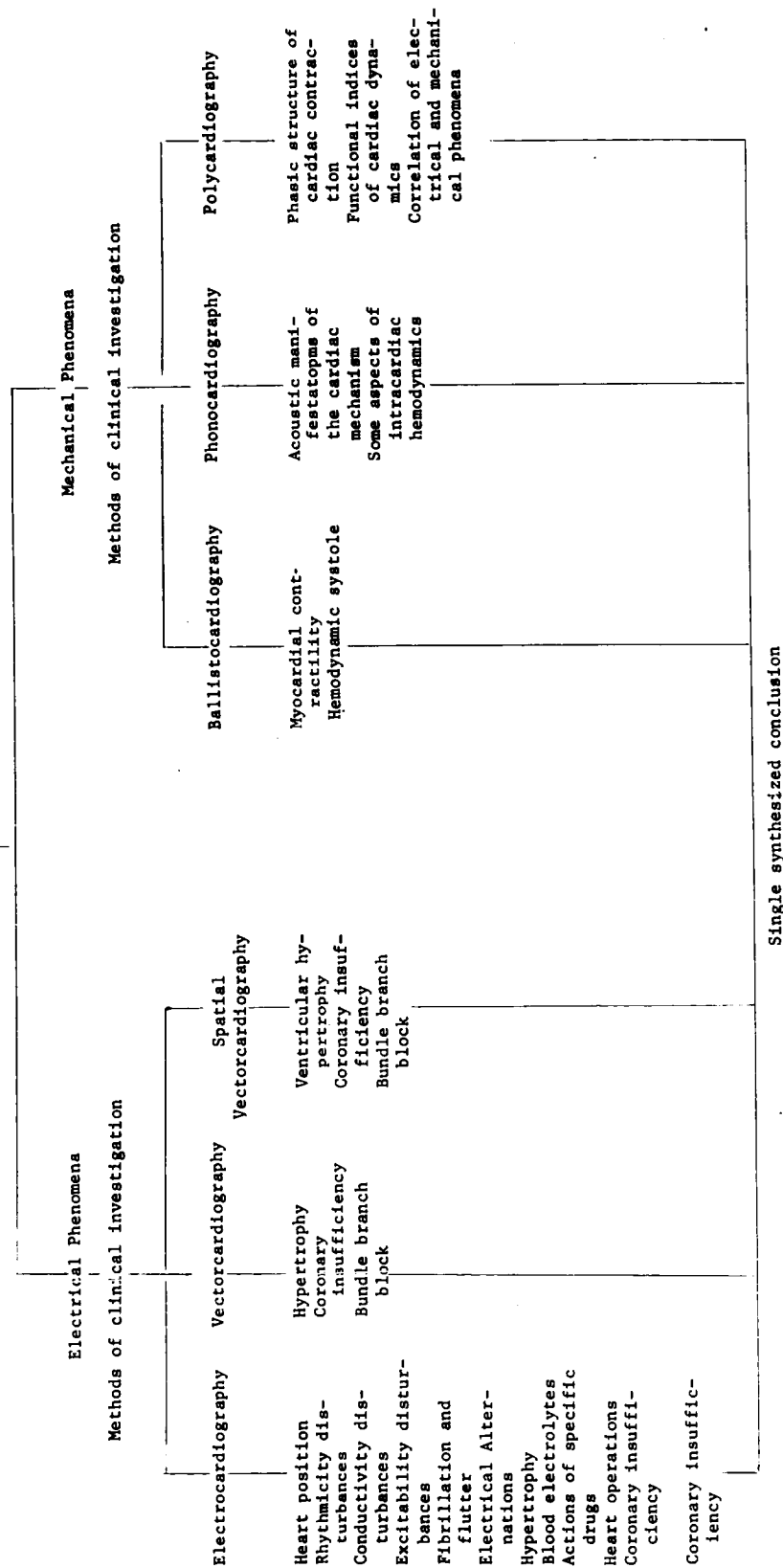
We present a diagram of the procedural method for synthetic electrocardiology which was worked out in our laboratory (page 39).

From this plan it is clear, that the presented methodology can give a clear and thorough description of the functional condition of the heart and can also contribute to the determination of anatomical diagnosis. Certainly, there exists a series of other electrocardiological methods, for example, dynamocardiography (Ye.B. Babskiy, 1957), mechanocardiography (N. N. Savitskiy, 1956), rheocardiography (Holzer, Polzer and Marko, 1945; Yu. T. Pushkar', 1961), kinetocardiography (Eddleman et al., 1953; L. B. Andreyev, 1961; I.Ye. /40 Oranskiy, 1961), cardiovibrography (Jackson, 1955), cardiocyclography (I. T. Akulinichev et al., 1960), cardiotoscopy (P. Z. Amirov, 1961), seismocardiography (B. S. Bozhenko, 1961), esophagocardiography (R. B. Minkin, 1961; and others). We have no experience of our own with these methods and are acquainted with them only through the literature. Although some of these methods can supplement the solution of certain problems, we did not find it necessary to introduce them into our plan, since our methodology gives a sufficiently complete answer to the demands which we set forth. In addition, for one specialist to cope with all these methods seems very difficult when, even without them, the methods of synthetic electrocardiology are complicated and very extensive.

We do not find it necessary to discuss further the fact that there exist other methods for the study of various functions of the heart, for example, such as roentgenokymography (Stumpf, 1951; and others), or roentgenoelectrokymography (Heckmann, 1959; V. V. Zaretskiy, 1957), or radiocardiography

*Here and subsequently by the word clinical we mean clinical electrocardiology.

CONTRACTION OF THE HEART



(Prinzmetal et al., 1948; Zacks, 1957; and others), however, these methods are not electrocardiological and therefore cannot be applied in our methodology.

PART TWO

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CLINICAL STUDY OF ELECTRICAL ACTIVITY OF THE HEART

CHAPTER I

ELECTROCARDIOGRAPHY

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I. INTRODUCTION

Electrocardiography is the oldest method and is also the basis of electrocardiology. Its development and refinement are inseparably tied with the progress of the electrophysiology of the heart. In 1843, Matteucci, in his letter to Humboldt, spoke of a "muscular current" in animal muscles. The presence of this phenomenon in the heart was verified by the well-known experiments of Kolliker and Muller in 1856: at each contraction of the heart there was registered a contraction of a neuro-muscular preparation of a frog, which was placed on the surface of the exposed heart. Using the capillary electrometer of Lippman, Waller in 1889, was the first to succeed in recording a human electrocardiogram, although the resulting curve did not present a clear pattern and could not serve as a basis for the broad development of electrocardiography. However, Waller's service lies in the fact that he showed the presence and distribution of isopotential lines on the surface of the human body.

In fact, the history of clinical electrocardiography begins in 1903 when the Dutch scholar Einthoven presented his original electrocardiograph. A rapid development of the new method followed, and very soon, thanks to the joint efforts of physiologists and clinicians, among whom the names of Einthoven, Lewis, A. F. Samoylov, V. F. Zelenin, Wenckebach, Rothberger, Wilson, and other authors should be especially emphasized, there was created all the necessary groundwork for the development of the science of electrocardiography, and its theoretical and practical problems. In the first period of development, this science occupied itself with the study of the disturbed rhythm of heart action, a little later with the working out of the problem of recognition of harmful myocardial and coronary insufficiency, and subsequently with the perfection of the method of recording and analyzing curves, and the introduction of a series of new leads (chest, unipolar), and an original vectorial principle of interpreting electrical phenomena of the heart mechanism. In order to imagine the feverish tempo of the development of electrocardiography, it is sufficient to recall that as recently as 1908, A. F. Samoylov expressed the hope that there would be found in clinical practice people with the physical and physiological training, and the good fortune to possess a method of recording the electrical currents of the heart, and that in 1914 there existed only 35 pieces of equipment in the entire world (Burch, 1961).

II. TECHNIQUES OF ELECTROCARDIOGRAPHY

All existing pieces of apparatus belong to one of two main types: current meters and voltage meters. A typical example of an apparatus of the first type is the electrocardiograph proposed by Einthoven with a filament galvanometer which has the following schematic construction. A fine metallic

filament with a diameter of no more than 2 or 3 microns is extended through a strong electromagnetic field. The ends of the filament are joined by metallic wires to the required points on the body of the subject, and with each heart contraction the resulting current passes through the filament, creating a corresponding magnetic field around it. Thus, in accordance with the physical law that movement occurs when two magnetic fields come into contact, there arises a movement of the filament, while the amplitude and direction of the movement is dependent on the nature of the flowing current. By means of a special optical system, consisting of a light source, a converging lens, and an illuminating microscope (placed inside one cylinder of the electromagnet), a bright beam of light falls on the center of the filament. Because of the projection microscope (placed in the second cylinder of the electromagnet) the enlarged image of the filament is projected on a twisting light sensitive band and in this way the oscillations of the filament are recorded on the band.

Another current meter, constructed on a similar principle, is the coil electrocardiograph, but here the filament is replaced by a light coil with a large number of loops. In this case there occurs a rotation of the coil, which is projected on to a light sensitive band by means of a system of "light source and mirror" (the mirror is glued onto the center of the coil).

The following principle of construction of electrocardiographs measuring voltage is very widely used today: by means of a tube amplifier, voltage, 145 on the order of a few millivolts, which originates upon the contraction of the heart, is multiplied 800 to 1000 times and is transmitted through the output tube of the anode current. The variation of the anode current, which is determined by the dynamics of the changes in voltage during heart contraction, is registered by means of a galvanometer. There also exists a voltage electrocardiograph which uses a cathode ray tube in which the amount of variation is registered on the screen of the apparatus (for details see page 168).

There are two methods of recording: photographic, where the variation is recorded on common film, and direct writing, where the variation is recorded directly on paper by means of various devices (ink writing, changing the color of special paper by the action of a heated pen, etc.). For the most part, the first method gives the truest value of the data obtained since the factors of inertia and friction, which are inherent in the operation of mechanical recorders, do not influence the nature of the curves obtained; the second method has a technical advantage in that it reduces the necessity of technical processing of the film, which is of increased value for emergency diagnosis and active investigation.

At the present time the multichanneled electrocardiograph is widely used in the simultaneous study of a series of leads; the advantage of such pieces of apparatus, especially in the solution of certain theoretical problems, is indisputable. The visual observation of the electrocardiogram on the screen of the cathode ray tube is becoming more and more widespread. This method is technically simple and is of great help in large scale investigations, and can be successfully used for teaching and continuous observation,

especially during surgical intervention. However, one visual observation alone does not have great scientific significance. In addition, it is undesirable for novice specialists to depend only on the data of such an observation. We find that the method discussed should be used only by experienced specialists.

All pieces of equipment have a special device for recording time since the interval between neighboring vertical lines can be 0.02, 0.04, or 0.05 sec. Each fifth vertical line is considerably thicker than the previous ones, so that between two neighboring thick lines the interval is 0.1 sec.; this method technically simplifies the numerical comparison of the length of time intervals. Some pieces of apparatus record a horizontal line with a distance between two neighboring lines of 1 to 2 mm. Modern equipment has a special device for obtaining various speeds of movement of the film or paper (12.5 mm./sec., 25 mm./sec., 50 mm./sec., 75 mm./sec., 100 mm./sec.). The factor of speed has technical significance in the analysis of the curve: the greater the speed of movement of the film, the further from each other will be the separate components of the electrocardiogram and the more accurate the various calculations. /46

The problem of standardization has great significance in electrocardiography. In order to make it possible to compare electrocardiograms photographed under various conditions or on various equipment, it has been assumed ever since the time of Einthoven that the apparatus is calibrated so that a change in voltage of 1 mv. (millivolt) causes a variation in the galvanometer.

It is necessary to remember various artifacts which arise as the result of contractions of skeletal muscles, polarization of the terminals, excess current in the room, and other causes.

III. ELECTROCARDIOGRAPHIC LEADS

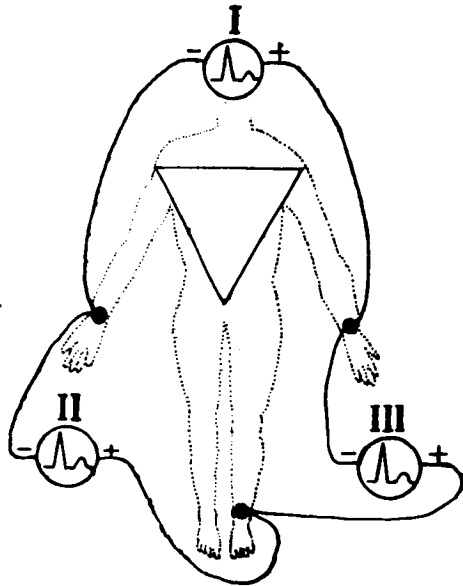
In the measurement of bioelectric potentials it is necessary to have contact between the measuring device and an electrolyte solution of the biological system (Kertis, 1956). In the clinical electrocardiograph, this is accomplished by placing metallic terminals on certain points of the human body and joining them to the apparatus by metallic wires. The electrodes must be made from nonpolarized metal, and, to establish good contact between the terminal and the skin, it is necessary to treat the skin beforehand with a saline solution or a paste of special composition. Such connection of two points of the body having different potentials is called a lead. In clinical practice indirect leads are used, i.e. the electrodes are not placed directly in the heart, but on the surface of the body; direct leads are used in rare cases, during operations on the heart.

All leads used in clinical electrocardiography may be classified according to two basic principles: in accordance with the placing of the terminals, the leads are classified into limb leads and chest leads; and in accordance with the physical characteristics, into bipolar and unipolar leads.

1. Bipolar Leads

As is already known from the first chapter, in this case both electrodes are active and actually record the difference in the potential between two points of the body.

A. Bipolar limb leads (Fig. 8) are also called standard or classical in honor of Einthoven who devised these leads. There are three such leads - first, second and third. They are indicated by Roman numerals.



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Fig. 8. Bipolar limb leads (standard leads). In the center is the Einthoven triangle.

a. Lead I. The terminal from the right arm is joined to the negative pole of the galvanometer of the apparatus, the electrode from the left arm to its positive pole. Thus, $I = VL - VR^*$.

b. Lead II. The terminal from the right arm is connected with the negative pole of the galvanometer, the terminal from the left leg to its positive pole. Thus, $II = VF - VR$.

c. Lead III. The terminal from the left arm is joined to the negative pole of the galvanometer, the terminal from the left leg to its positive pole. Thus, $III = VF - VL$.

*In accordance with international nomenclature, the potential of the right arm is marked VR, of the left arm VL, of the left leg VF from the first letters of the English words, voltage, right, left, foot. The corresponding Russian designations are P, L, N, from the first letters of the words pravaya, levaya, noga.

As is seen in figure 8, in these leads the terminals are placed at a definite distance, approximately equidistant from the heart. However, in reality, the limbs only play the role of conductor, and everything occurs just as if the terminals were placed on the torso at the junction of the limbs. When these points are joined, an equilateral triangle is formed with the heart at its center. The meaning of this triangle, called the Einthoven triangle, and the theory of Einthoven which is based on it, will be discussed in detail (see page 66 orig. text).

B. Bipolar chest leads were brought into use later, when certain inadequacies of standard leads became apparent. Thus, the standard leads are located at a certain distance from the heart and therefore the potential recorded by them is less than that which actually arises during stimulation of the heart. The standard leads cannot give information about the electrical condition of the separate parts of the heart, since they record only the difference of potential between two points distant from the heart. In addition, the standard leads cannot show those electrical energies of the heart which /48 are in a direction perpendicular to the frontal plane of the Einthoven triangle.

In the case of the chest lead, one terminal is placed at a given point of the chest wall and another on one of the extremities; the curve thus obtained is basically a reflection of the potential of the chest terminal, which, due to its proximity to the heart, is able to pick up the maximum potential, while the distant terminal, located at a relatively large distance from the heart, will record the minimum potential. In this way, with the help of the chest leads, it is possible, to a certain extent, to gain information about the electrical phenomena actually occurring in definite parts of the heart. The chest terminal is called test or active and the distant terminal is called neutral or passive.

However, as Sodi-Pallares (1956) demonstrates, the potential of the active terminal is considerably less than the potential of the actual voltage of the corresponding part of the heart, so much so, that it even may be equal to the potential of the distant terminal. As a result, in the case of the chest lead we are dealing with the conventional bipolar lead (although Bayley, 1943, calls it a half pole lead), which records the difference in the potentials between two points. Therefore, the curve obtained cannot be considered a complete picture of those electrical phenomena which occur in the part of the heart lying under the terminal.

For positioning of the test terminal, the following points exist on the chest wall, (they are designated by the letter C from the English word "chest", and in Russian nomenclature, by G from the word "grud'", and the location of the terminal is denoted by Arabic numerals):

- C₁ - by the right edge of the sternum in the fourth intercostal space;
- C₂ - by the left edge of the sternum in the fourth intercostal space;
- C₃ - on the left peristernal line at the point of its intersection with

the line joining C_2 and C_4 ;

C_4 - on the left central scapular line in the fifth intercostal space;

C_5 - on the left front axillary line, on the level of the horizontal line which passes through C_4 perpendicular to the axillary lines;

C_6 - on the left central axillary line on level with the same horizontal line.

In clinical practice the above-mentioned 6 chest points are the ones used most. In some cases the following chest points are also used:

C_7 - on the left rear axillary line on a level with the same horizontal /49 line;

C_8 - on the left scapular line on a level with the same horizontal line;

C_9 - on the left perivertebral line on a level with the same horizontal line.

For special investigations, certain positions on the right half of the chest wall are used. These points have a symmetrical distribution with respect to the left points and are signified by C_3R , C_4R , etc. Sometimes the

chest terminal is placed in the region of the xiphoid process; this position is signified by CE.

In recording the chest leads, if the passive terminal is placed on the right arm, the lead is marked CR (GP in Russian nomenclature), if it is placed on the left arm, the lead is marked CL (GL), and if the terminal is located on the left leg, the lead is marked CF (GN). Until the introduction of single pole leads, the chest lead CR was used most frequently in clinical practice.

2. Unipolar Leads

Unipolar leads have definite advantages since they provide the possibility of explaining complicated electrocardiographical phenomena by simple physiological concepts, and create the theoretical background for understanding the substance of various electrocardiographical descriptions (Lipman and Massie, 1956). A unipolar recording may be obtained in the case where one of the electrodes is completely indifferent, i.e. its potential is zero or nearly zero. Wilson (1933) developed the technique of the single pole lead by means of the central terminal or, as some authors call it, the universal passive terminal. In this device (Fig. 9), the wires from the three extremities are joined to a single point and in each wire there is a resistance of 5000 ohm. Mathematical calculations and also the empirical observations of various authors show that the potential of the central terminal (VT) is equal /50 to zero. This may be proved in the following manner.

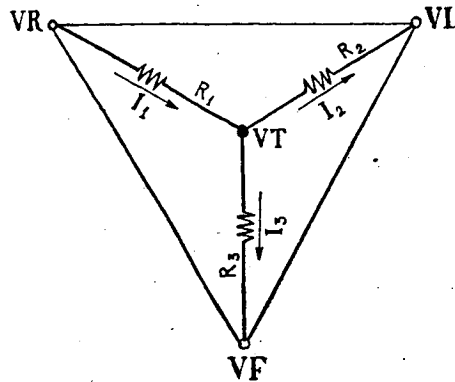


Fig. 9. Zero potential of the central terminal. R - resistance, I - strength of current, the sign of resistance in each wire corresponds to 5000 ohms (explanation in text).

As is seen in Fig. 9, the heart current from the body enters the central terminal through the right arm (I_1), and from there reenters the organism through the left arm and leg (I_2 and I_3). From the first law of Kirkhof it is known that the sum of the intensities of the currents flowing toward any point of branching of conductors is equal to the sum of the intensities of the currents flowing away from it. Thus,

$$I_1 = I_2 + I_3. \quad (1)$$

In accordance with Ohm's law, the intensity of a current passing along a conductor is directly proportional to the difference in the potentials (electromotive force) and inversely proportional to the total resistance. Thus, in the present case

$$I_1 = (VR - VT)/R_1, \quad I_2 = (VT - VL)/R_2, \quad I_3 = (VT - VF)/R_3 \quad (2)$$

Substituting (2) in (1), we obtain,

$$(VR - VT)/R_1 = (VT - VL)/R_2 + (VT - VF)/R_3 \quad (3)$$

but since $R_1 = R_2 + R_3$, it follows that,

$$VT = (VR + VL + VF)/3 \quad (4)$$

Considering that Einthoven's triangle, with the three angles R, L and F, is equilateral, and that the dipole of the heart is in its center, we may conclude that

$$VR + VL + VF = 0 \quad (5)$$

consequently,

$$VT = 0.$$

(6)

There is considerable difference of opinion in the literature with regard to the zero potential of the central electrode. Simply the fact that Einthoven's triangle is not always equilateral and the dipole of the heart is not located in its center, causes doubt in regard to the accuracy of the stated proposition. According to Osborne and Dower, (1957), the use of the central electrode sometimes leads to inaccuracies in diagnosis. D. F. Presnyakov (1959) in general does not consider this lead to be unipolar. Nevertheless, the magnitude of the potential of the central electrode is insignificant and for all practical purposes it is the same in all cases. Thus, according to the data of Dower, Osborne and Moore (1959), the potential of the central electrode exceeded 0.3 mv. in only 8% of the cases studied. Considering all these opinions, we are in agreement with the opinion of M. B. /51 Tartakovsky (1958) that the central electrode of Wilson is the most expedient of all the indifferent electrodes used at the present time.

A. Unipolar limb leads. In these leads the central electrode is used as the indifferent electrode, and the exploring electrode is placed on the corresponding extremity. Thus, the unipolar lead from the right arm is marked VR, from the left arm VL and from the left leg VF (Fig. 10); the corresponding Russian designations are P, L, N.

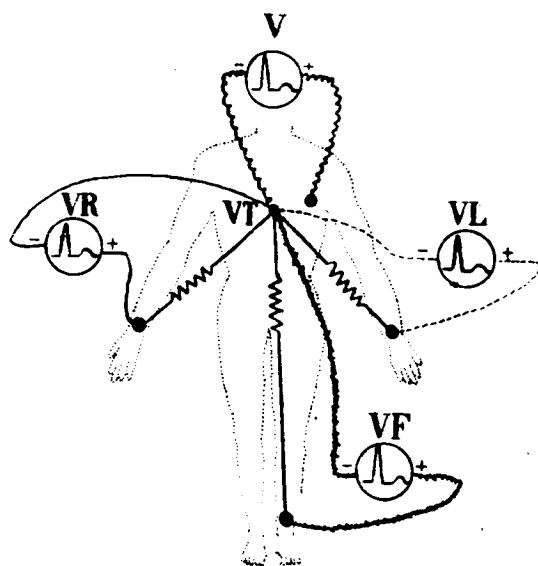


Fig. 10. Unipolar limb leads.

Goldberger (1942), proceeding from the fact that the same shape of curve is obtained even without the inclusion of the above-mentioned resistances, proposed an indifferent electrode of three wires from the limbs. However, further observation showed that, with the application of such an indifferent electrode, the need arose for a galvanometer that was 1.5 to 2 times as sensitive, because of a reduction in the amplitude of the waves in the

curve obtained. The work of Goldberger proved that, without influencing the shape of the curve, it was possible to increase the voltage by making a central indifferent electrode from the junction of the leads of only two electrodes, and the lead from the third limb, which is the subject of investigation in the present case. This electrode then is joined to the second positive electrode of the galvanometer. These leads are called increased or augmented, and are designated by the letter "a" (from the English word "augmented") and in Russian nomenclature by the letter "u" from the word "usilennoye". Thus, the augmented single pole lead from the right arm is designed aVR (uP), from the left arm aVL (uL), and from the left leg aVF (uN) (Fig. 11).

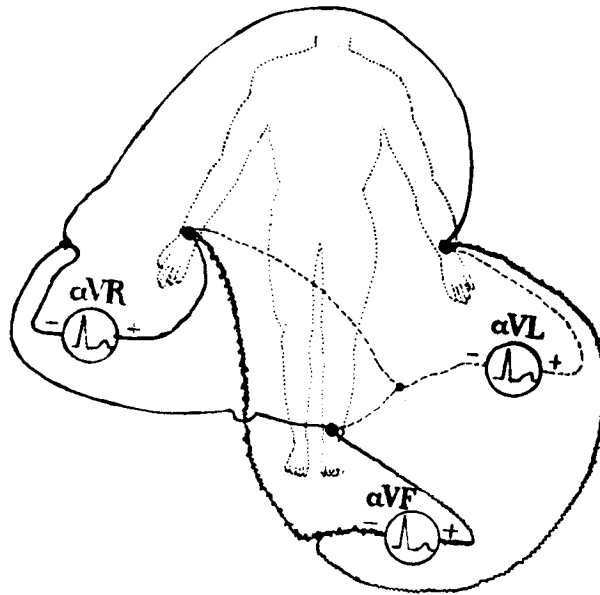


Fig. 11. Augmented unipolar limb leads.

It has been exactly calculated that the voltage of the augmented limb leads is 50% greater than the voltage of the non-augmented leads. This statement, for example in reference to lead aVR, can be proved in the following way. In this case the potential of the central electrode is not zero, but represents the average value of the potentials of the two limbs of which it consists, i.e. in this case

$$VT = (VL + VF) / 2. \quad (1)$$

It is known that

$$aVR = VR - VT \quad (2)$$

thus, proceeding from (1), we may write

$$aVR = VR - (VL + VF) / 2. \quad (3)$$

It has already been proved that

$$VR + VL + VF = 0 \quad (4)$$

or

$$VL + VF = - VR. \quad (5)$$

Now (3) may be written

$$aVR = VR - (-VR)/2 \quad (6) \quad \underline{/53}$$

i. e.

$$aVR = 3VR/2. \quad (7)$$

The use of the augmented leads has been greatly extended and, as will be seen later, in certain cases has an advantage over the non-augmented. These **two types** of unipolar limb leads have a definite relationship to the standard leads. It may be stated, in general, that lead I corresponds to lead aVL or in the reverse pattern to lead aVR; lead II corresponds to lead aVF or in the reverse pattern to aVR; lead III corresponds to lead aVF or in the reverse pattern to lead aVL. In addition, it is possible to establish mathematically the value of the potential at any angle of Einthoven's triangle (Wilson, Macleod and Barker, 1931). It has been calculated that $VR = (-I - II)/3$, $VL = (I - III)/3$, $VF = (II + III)/3$ in regard to the augmented leads,

$$aVR = (-I - II)/2, \quad aVL = (I - III)/2, \quad aVF = (II + III)/2.$$

These relationships arise from the following mathematical calculations. We take for example leads VF and aVF.

$$\text{It is known that: } I = VL - VR, \quad II = VF - VR, \quad III = VF - VL, \quad (1)$$

$$\text{then } II + III = (VF - VR) + (VF - VL) = 2VF - VR - VL; \quad (2)$$

$$\text{in addition } VR + VL + VF = 0 \quad (3)$$

$$\text{or } VF = -VR - VL. \quad (4)$$

$$\text{Substituting (4) into (2) } II + III = 2VF - (-VF) = 3VF, \quad (5)$$

$$\text{thus, } VF = (II + III)/3. \quad (6)$$

$$\text{Since } aVF = 3VF/2 \quad (7)$$

$$\text{it follows that } aVF = (II + III)/2. \quad (8)$$

B. Unipolar chest leads. Wilson's central electrode is used with these leads and the exploring electrode is placed on the usual chest points. /54

These pericardial leads* are designed V_1 , V_2 , etc., (in Russian nomenclature GO_1 , GO_2 , etc., from the word "ob'edinenny"). The resultant pattern

in this case is the true reflection of the potentials of those parts of the heart which are located under the exploring electrode (more exactly, the unipolar electrode registers not the net local potential of the part of the heart corresponding to it, but the total potential of the vectors of the heart, which appear in the given part of the heart). Thus, the leads V_1 and V_2 indicate the electrical condition of the right heart, V_5 and V_6

represent the electrical processes in the lateral partition of the left ventricle, and V_3 and, in part, V_4 correspond to the intermediate zone and re-

cord electrical phenomena in the area of the partition between the ventricles. We are in agreement with Sodi-Pallares (1956), that this does not always correspond exactly, and such factors as the position of the heart and the change in the area of the designated parts of the heart, may have an influence on the shape of the curve obtained. Nevertheless, as has been shown (Simonson, Schmitt and Nakagawa, 1959), it is no longer possible to argue about which of the chest leads gives the best results, and the advantages of the pericardial leads are almost universally accepted.

In our clinical practice we use 12 leads: three standard leads, three augmented unipolar limb leads and six pericardial leads (V_{1-6}). We find

that only such a detailed recording of the electrocardiogram can give a many-sided representation of the electric condition of the heart and it is impossible to agree with the supposition (Schaffer et al., 1956) that instead of six limb leads, only leads I and aVF need to be recorded.

3. Special Leads

These leads are not used in everyday clinical practice, but serve only certain investigatory goals.

A. Modified chest leads. Nehb (1938) proposes a system of bipolar chest leads in which the electrode from the right arm is placed in the second intercostal space to the right of the sternum, the electrode from the left arm is placed in the area of the rear axillary line on a level with the apex of the heart, and the electrode from the left leg in the area of the apex of the heart. The author considers that the resulting "small triangle of the heart" can record the front, rear and lower potentials of the heart. R. P. Stamboltsyan (1962) proposes bipolar chest leads in which the indifferent electrode from the right arm is placed in the third intercostal space by the right edge of the sternum, and the exploring electrode at the usual six chest points. These leads, which are designated CS, acquire significance in the

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*The expression pericardial leads in the future will signify unipolar chest leads, to distinguish them from the conventional bipolar chest leads.

diagnosis of coronary insufficiency and posterior infarctions (A. T. Simonyan, R. P. Stamboltsyan and Ye.N. Apinyan, 1958).

Cossio and Bibiloni, (1956) propose three horizontal bipolar leads (placed in one common horizontal plane), which, at the level of the point of zero potential of the heart (see page 167) forms an equilateral triangle with the apex at the front and the base at the back. The unusual distribution of the terminals, because of the resulting rear, front and septal leads, designated by the letters A, B, C, is described by Trethewie, (1953, 1958). He found that these leads have definite advantages over the system of 12 leads in discovering and determining the location of an infarct, but the data of Lancaster, Semple and Kelly, (1960) refute his opinion.

B. Esophageal leads. A small metallic globe is inserted into the esophagus with the help of a probe and joined to the wire of the electrocardiograph and the electrocardiogram is recorded at various levels. Thus, at a depth of 35 cm. from the front teeth, the pattern above the auricle is studied, at a distance of 35 to 42 cm. the pattern of the auricle is recorded, and at a distance of 42 cm. the operation of the ventricles is studied. In these cases the rear wall of the heart is principally studied while, at the same time, the auricular T waves are very well defined (V. I. Maslyuk, 1955, 1958; Brody and Copeland, 1959; Rubin, Jagendorf and Goldberg, 1959).

C. Endocardial leads. By means of a special catheter, the exploring electrode is inserted into the heart at the time of its probing, and the electrical phenomena of the cavity of the auricles and ventricles are recorded. Luisada and Liu, (1957) propose that the exploring electrode be a 5% saline solution with 10% heparin inside the catheter. The technique of endocardial electrocardiography was developed in connection with improvements in heart surgery and plays a large role in the solution of certain theoretical and practical problems.

D. Electrocardiography of the fetus (Fig. 103A). According to the data of various authors, such an investigation acquires great significance in certain clinical situations. In 1938, Strassman and Mussey proposed a simple method which uses the five conventional bipolar limb leads from the extremities. Southern (1954, 1957), proposes an apparatus and his own system of leads which has certain advantages. Larks and Dasgupta (1958) designate four leads for various periods of pregnancy, three of which are bipolar (central, longitudinal and front to back), and one is unipolar.

IV. NORMAL ELECTROCARDIOGRAMS

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The electrocardiogram is the total curve of those electrical phenomena which arise during heart contraction, or more accurately, at the time of heart stimulation.

In the electrocardiogram there are distinguishable waves, segments and complexes. Each electrocardiographic wave is a deflection from the iso-

electric line and has its beginning and end: the beginning of the wave corresponds to that point at which it deflects from the isoelectric line, and the end to that point on the isoelectric line to which it returns after forming the defined pattern. The wave is positive if it deflects upward from the isoelectric line and negative if it deflects downward. In addition, it can be diphasic, with an initial positive deflection and a secondary negative deflection (+ -) or conversely, with an initial negative deflection and a second positive deflection (- +). Each wave has its own particular shape and the amplitude or magnitude of the waves is determined by the distance in mm. from its apex to the isoelectric line, and its length or breadth is measured by the interval of time, in seconds, between the beginning and the end of the wave.

A segment consists of the isoelectric period and corresponds to that phase of heart activity which is not accompanied by the appearance of electromotive force. Each segment has a definite length and form.

If the wave or segment corresponds to some definite moment of excitation of the heart, then the complex is the reflection of one entire phenomenon, which is included in this complicated process, and therefore it may include within itself some waves and segments.

In the normal electrocardiogram six waves, three segments and two complexes are found (Fig. 12).

Since 1885, in accordance with the suggestion of Einthoven, the waves of the electrocardiogram are provisionally designated by the letters of the Latin alphabet P, Q, R, S and T. Of these waves, P, R and T are positive and are necessary waves for a normal electrocardiogram, and Q and S are negative and may be absent in a normal electrocardiogram. The U wave is positive and is seldom encountered.

The segment P - Q (or α) falls between the end of the P wave and the beginning of the Q wave; in the absence of the Q wave we have the segment P - R. Segment S - T is located between the end of the S wave and the beginning of the T wave; in the absence of the S wave we have the segment R - T. In practical work this segment may be simply called segment RS - T. Segment T - P falls between the end of the T wave and the beginning of the P wave of the following heart cycle.

The auricular complex basically consists of the P wave (see below), and 57 the ventricular complex corresponds to the interval QRST or Q - T, which may be divided into three constituent parts: the first part is the complex QRS, the intermediate part is the segment RS - T and the final part is the T wave.

To denote all the designated waves, segments and complexes, Briskier (1956) proposed a special system in which the letters are replaced with colors, but we consider it easier and better to adhere to the universally accepted system.

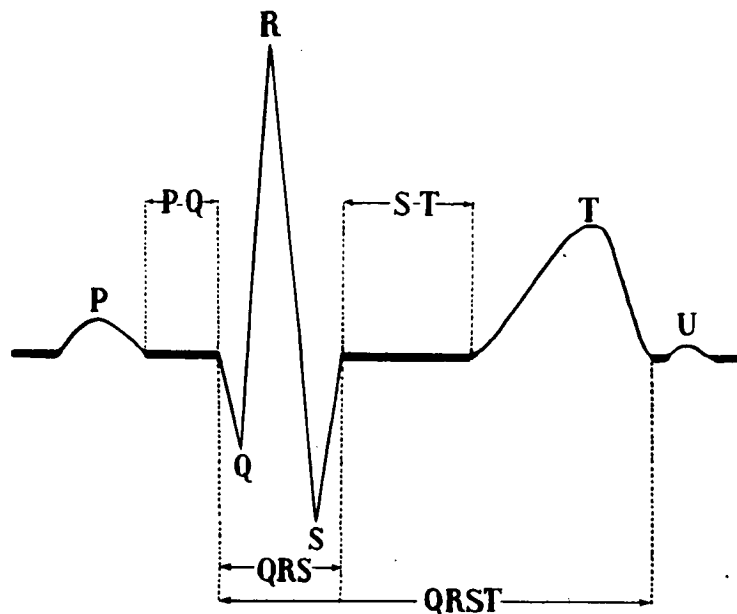


Fig. 12. The normal electrocardiogram (schematic).

1. The Auricular Complex

The stimulation of the auricles is reflected in, and consists of the P wave, the segment P - Ta and the Ta wave (the letter "a" from the English word atrial), in which these three components correspond to the process of depolarization, the voltage phase and the process of repolarization of the auricles. In the normal electrocardiogram only the P wave of the atrial complex is expressed, and the segment P - Ta and the Ta wave blend into the components of the electrocardiogram following the P wave; the behavior of segment P - Ta may sometimes resemble the situation of segment P - Q. In certain cases when there is expressed retardation of atrioventricular conductivity, it shows up clearly in segment P - Ta and the Ta wave.

The P wave arises as the result of the algebraic sum of electrical phenomena from the excitation of the right and left auricles, the first half of the wave corresponding principally to the excitation of the right auricle and the second half to the excitation of the left auricle. Observations have been made, according to which the excitation of the right auricle is expressed by the appearance of a positive wave, and excitation of the left auricle by a negative wave; but in a normal electrocardiogram, the P wave is positive because in the right auricle the excitation process sets in somewhat earlier than in the left and, as a result of this, the potential of the right auricle dominates the total pattern.

The morphology of the normal P wave consists of an open upward deflection with a rounded or sometimes slightly pointed apex and a symmetrical open downward deflection. Its amplitude varies from 0.5 mm. to 2.5 mm.; the length is 0.1 sec.

To characterize the P wave, Marcruz, Perloff and Case (1958) propose to define the coefficient, length of P wave/length of segment P - Q, which as a rule is from 1.0 to 1.6 mm. In the presence of pointed P waves, Gross (1961), proposes to form a triangle, taking as the base the isoelectric line, and to define such indicators as the area of the wave (height x length ÷ 2, in microvolt-sec. mm.), the time of ascent (the length of projection of the upward deflection from the isoelectric line in percent, with respect to the length of the P wave), the velocity of ascent (amplitude of wave P/time of ascent) and the three angles of the triangle.

Normally, the segment P - Ta is a little lower than the isoelectric line; wave Ta is turned opposite to the direction of the P wave, it is small, and its amplitude depends on the magnitude of the P wave.

Thus, the atrial complex indicates the electrical systole of the auricles, the length of which varies depending upon the atrial rhythm, from 0.19 to 0.53 sec.; the more frequent the rhythm of contraction of the auricles, the less the length, and vice-versa (Kesselman et al., 1956).

2. The Ventricular Complex

The process reflects the extension of the excitation wave into the ventricles and is the result of the algebraic sum of the excitation of both ventricles. Some earlier authors considered that the R wave corresponded to the excitation of the right ventricle and that the S wave expressed the excitation of the left ventricle, or otherwise, that complex QRS reflected the excitation process, and the T wave the contracting. At the present time it must be considered as established that the beginning part of the ventricular complex corresponds to the process of depolarization of the ventricles, the intermediate part is the voltage phase, and the final part is the process of repolarization. However, this cannot explain why the phases of depolarization and repolarization of the excitation of the ventricles cause waves of the same direction (the largest wave of complex QRS and wave T are positive), because when an isolated muscle bundle is stimulated a curve is recorded with two oppositely directed phases. Various theories have been proposed to explain this phenomenon. A group of authors proceeds from the supposition of German investigators that the normal ventricular complex is formed by the interaction of the partial curves of the right and the left ventricles (Fig. 13): the partial pattern of the right ventricle is characterized by the first positive phase and the second negative phase, and the partial curve of the left ventricle has a converse configuration. These authors propose that in the normal electrocardiogram the first phase is positive because the right ventricle is stimulated a bit earlier than the left and dominates its partial pattern. The positiveness of the T wave is explained by the fact that the second phase of the partial curve of the right ventricle has comparatively less amplitude than the corresponding phase of the left partial curve, and in their interaction, the pattern of the left ventricle dominates. This theory of the interference of the left and right ventricle currents was held by A. F. Samoylov, (1908), and V. F. Zelenin, (1913).

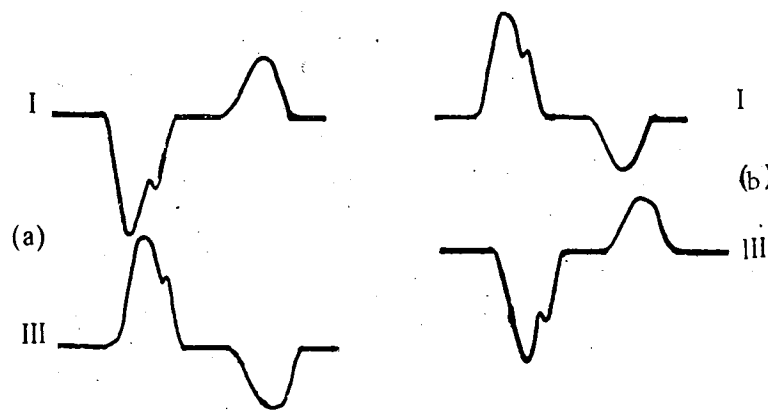


Fig. 13. Partial electrocardiograms (schematic): (a) left ventricle; (b) right ventricle.

Craib (according to Sigler, 1946) considers that the QRS complex is recorded before the appearance of any mechanical movement of the myocardium, and that the T wave is inscribed at the time of heart contraction. From this they derive the hypothesis that, as a result of heart contraction, the direction of extension of impulses changes in such a way that the second phase becomes positive. /60

We find the following point of view more acceptable. The process of stimulation in the ventricles extends from their endocardial surface toward the epicardial, but since the subendocardial layer remains in an excited condition longer than the subepicardial, the process of repolarization begins earlier in the subepicardial layers and extends from the outside inward, i.e. in a direction opposite to the process of depolarization. Consequently, in agreement with the modern concept of the significance of the direction of these processes (see page 32), the second phase takes the same direction as the first. It is necessary also to remember that the process of repolarization is distinguished from the process of depolarization by its slower development, and it therefore gives another configuration to the T wave. From these points we may evaluate the opinion of A. F. Samoylov, that the T wave gives a representation of the functional condition of the heart, because it is well known that repolarization is a labile process and quickly changes under the influence of even ordinary physiological factors.

With regard to the genesis of the U wave, there is much controversy, and it even became the subject of a special symposium (1957). Lepeschkin, (1951, 1957) gives much space to this problem; Lepeschkin and Surawicz (1953) find that the U wave occurs in almost all subjects. Some authors propose that the U wave arises as a result of the lengthening of the action potential in some part of the myocardium of the ventricle, or is the potential of after-effect, or else the reflection of the stimulation of the main vessels. Carouso, Lenegre and Chevalier (1954) attach particular significance to the delayed repolarization of the separate parts of the interventricular septum; Furbetta et al. (1957) consider the U wave to be the reflection of the potential of the papillary muscles, and Holzmann (1957), proposes that it is caused by

the position of the walls of the ventricle during the early phase of diastole.

The morphology of the normal QRST complex is characterized in the following manner. The Q wave drops sharply and forms a pointed apex, after which it rises sharply and merges with the beginning of the R wave. Its amplitude on the average is 3-4 mm. and its interval is 0.03 sec.

The R wave consists of a sharply ascending deflection with a pointed apex and a less sharply descending deflection. The amplitude of this largest wave of the ventricular complex varies within broad limits, from 4 to 25 mm., and in interval, from 0.05 to 0.08 sec.

The S wave is recorded directly after the R wave, it drops sharply forming a pointed end and rises less sharply toward the isoelectric line. On the average its amplitude is 6 mm. and the interval is 0.03 to 0.04 sec. /61

In this way, the interval QRS normally varies in the range of 0.10 sec. For comparison of amplitude characteristics of the individual waves of this complex, the waves with small amplitude are designated by lower case letters, and the waves with large amplitude by capital letters. Thus, the description of a normal pattern may be marked qRs; if Q is absent, and R has a small amplitude and S is large, it may be written rS, etc.

Segment RS-T is located at the level of the isoelectric line (this level is defined with the help of segment P-Q). Its period is dependent upon a series of factors, in particular on the rhythm of contraction of the heart; in certain cases the segment RS-T may be absent, then we speak of the junction RS-T.

The T wave is formed by a slow upward deflection (with some protuberance), a rounded apex and a rather sharp downward deflection, which causes its normal configuration to be asymmetrical, the apex of the wave is located nearer its end than its beginning. The shape and amplitude of the T wave varies significantly: the amplitude on the average is equal to 1.5 to 7 mm. and the interval to 0.05 to 0.25 sec. (according to L. I. Fogelson, 1957). There is definite significance in the quantitative and qualitative relationships of the waves R and T, depending, according to Gross (1956), on the size of the angle between the downward deflection of the T wave and the isoelectric line, and also on the form of the intersection of the R wave with the straight line running from the downward deflection of the T wave toward the R wave.

The U wave, which usually appears 0.04 sec. after the end of the T wave, has a sharply rising deflection, a rounded or, less frequently, pointed apex and a less sharp downward deflection. It is small and often appears with a slow rhythm which does not exceed 100 beats a minute (Nahum and Hoff, 1939).

In this way, the Q-T interval represents the electrical systole of the ventricles, and its interval depends on the rhythm of heart contraction. For determining the normal duration of the electric systole at varying frequencies of heart rhythm, a series of formulas is proposed. We base our

work on the formula of Bazett (1918):

$$Q - T = K \sqrt{c},$$

where K is a constant value (0.37 for men and 0.40 for women), and c represents the duration of one heart cycle (which is defined as the interval R-R on the electrocardiogram).

L. I. Fogelson and A. I. Chernogorov (1927), propose to define the systolic index which expresses the relationship of the duration of the electric

systole to the interval of the heart cycle in percent $\left(\frac{Q - T}{R - R} \times 100\%\right)$. We /62

attach more significance to the determination of the absolute value of the electric systole, but from time to time we also use the systolic index.

Taran and Szilagy (1947, 1951), propose to define a corrective factor Q-T which is equal to $\frac{Q - T}{\sqrt{c}}$; according to their data, and also according to the data of other authors (M. N. Podnos, 1961), this index has definite advantages.

3. Five Basic Forms of the Ventricular Complex

The form of the ventricular complex significantly changes depending on the part of the heart it reflects. In this problem we base our work on the data of Goldberger (1954), who distinguishes the following five basic forms of the normal ventricular complex (for a better understanding of these forms see the section "Electric activity of the heart".)

1. Complexes of the type qR or qRs with a positive T wave are characteristic for a unipolar lead from the epicardial surface of the left ventricle (Fig. 14a). In this case the terminal of the unipolar lead is also placed on the left side of the interventricular septum and on the endocardial surface of the right ventricle. The initial q is formed as a result of the original small vector going from left to right, i.e. further from the test terminal. A little later the stimulation of the left ventricle starts from the inside outward, i.e. in the direction toward the terminal, and therefore the terminal records R; the simultaneous stimulation of the right ventricle causes an electrical force further from the electrode but due to the large muscle mass of the left ventricle its force dominates in the final pattern. However, in view of the fact that the individual parts of the right ventricle are stimulated later than the left ventricle, they may sometime cause s. Such complexes are often encountered in leads V_5 , V_6

and, depending on the position of the heart, in leads aVL and aVF.

2. Complexes of the type rS, rSr' or RS with the usual positive T wave are characteristic for a unipolar lead from the epicardial surface of the right ventricle (14b). In this case the exploring electrode is placed also

on the right side of the interventricular septum and the endocardial surface of the left ventricle. The initial r is formed as a result of the fact that the original vector is directed toward the side of the electrode. A bit later the stimulation of the right ventricle initiates forces directed toward the /63 side of the electrode, but they turn out to be weaker than the force of stimulation of the left ventricle which passes even further from the electrode. The result of this interaction of forces is the appearance of the S wave. The final wave r' which is sometimes observed is explained by the delayed stimulation of the individual parts of the right ventricle. These complexes occur in leads V_1 , V_2 , V_3 and, depending on the position of the heart, in leads aVR, aVL, aVF.

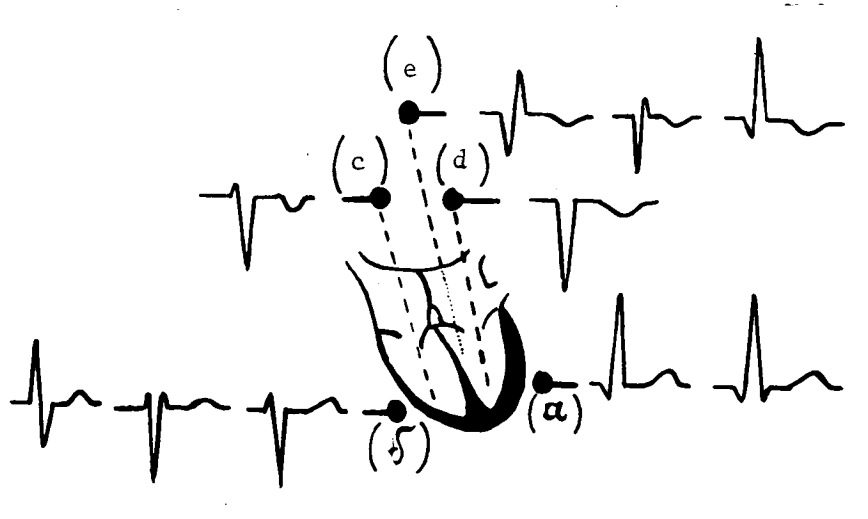


Fig. 14. The five basic forms of the ventricular complex: (a) from the epicardial surface of the left ventricle; (b) from the epicardial surface of the right ventricle; (c) from the cavity of the right ventricle; (d) from the cavity of the left ventricle; (e) from the rear surface of the heart.

3. Complexes of the type rS with a negative T wave are characteristic for a unipolar lead which is applied to the side of the right ventricular cavity (Fig. 14c). The initial r wave arises as a result of the fact that the original vector is directed toward the electrode. During the stimulation of the ventricles, the right as well as the left, the process is directed further from the electrode, thus the S wave is obtained. Depending on the position of the heart, this complex may be observed in the most diverse leads, but especially in lead aVR.

4. Complexes of the type QS with a negative T wave are characteristic for a unipolar lead which is placed on the side of the left ventricular cavity (Fig. 14d). The complete negative complex is explained by the fact that the original vector and the stimulation of both ventricles is directed further from the electrode. Depending on the position of the heart, this complex may be observed in leads aVR, aVL, or in the posterior superior sections.

5. The complexes of the type QR, Qr, or qR with negative T waves are characteristic for a unipolar lead which is placed in the region of the rear surface of the heart (Fig. 14e). This electrode, through the left auricle, is placed on the side of the left ventricular cavity, the stimulation of which causes forces further from the electrode, and the appearance of the Q wave. But during the stimulation of the rear surface of the left ventricle the electrical forces extend in the direction of the electrode and the R wave is recorded. These complexes, depending on the position of the heart, can be observed in leads aVR, aVL, aVF and others.

/64

This classification of the types of ventricular complexes has great practical significance. It is true that it may turn out to be a bit schematic, conditional and not fully justified in theory, but nevertheless it is of great assistance in our daily work.

4. The Time of Onset of Intrinsicoid Deflection

The electric forces which govern the appearance of electrocardiographic variation when the recording electrode is placed on the surface of the exposed heart, arise from two sources: the depolarization of the parts which lie directly under the exploring electrode, and the stimulation of the other parts, located around the place where the electrodes are positioned; Lewis, (1925), called them respectively intrinsic and extrinsic variations. However, such an analysis is possible only in the case of fixed leads, which are not used clinically. Proceeding from this, and on the basis of the data of Macleod, Wilson and Barker, (1930), on intrinsicoid deflection in the pericardial leads, it was proposed to define, for clinical use of pericardial leads, the time of onset of intrinsicoid deflection, or, as it is called by M. B. Tartakovskiy (1958), the time of local negative potential. Although it may be disputed, in clinical electrocardiography it is assumed that the internal variation begins from the maximum point of the positive wave of the ventricular complex in the pericardial leads, i.e. from the apex of the R wave. Consequently, the interval of time from the beginning of complex QRS to the apex of the R wave is the time required for the appearance of the stimulation process in the part of the myocardium lying under the electrode, i.e. the time for the onset of intrinsicoid deflection. Under normal conditions it is equal to 0.02-0.025 sec. in leads V_1 , V_2 , and 0.04-0.05

sec. in leads V_5 , V_6 ; the difference between these chest positions is explained by the fact that the wall of the left ventricle is thicker than the wall of the right and, in addition, the right ventricle enters into the stimulation process a bit earlier than the left.

V. ANATOMICAL AND ELECTRICAL POSITIONS OF THE HEART

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In the normal anatomical position of the heart in the thorax, the right ventricle occupies, basically, the right front surface of the heart, and the left ventricle, its left side and rear surface; the long axis from the base (left auricle) to the top (left ventricle) of the heart is directed forward, downward and to the left. However, in connection with various

physiological and pathological factors, significant changes in the position of the heart may be observed; they arise as the result of the rotation of the heart about its three main axes.

1. The antero-posterior axis passes through the center of the heart, and rotation clockwise leads to a vertical position, and rotation counter-clockwise gives it a horizontal position (Fig. 15a).

2. The longitudinal axis is directed from the center of the base of the heart toward its top. During clockwise rotation about this axis (the investigator looks downward along the heart from its top) the right ventricle occupies all the front surface and the left ventricle slips back and down. During counterclockwise rotation the left ventricle occupies a large part of the front surface of the heart (Fig. 15b).

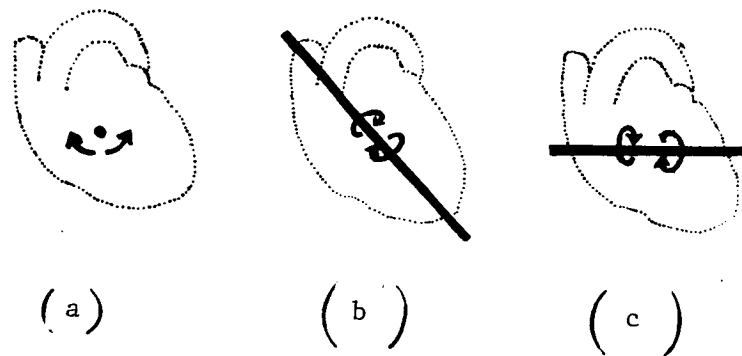


Fig. 15. Rotations of the heart about its three basic axes; (a) rotations about the antero-posterior axis; (b) rotations about the longitudinal axis; (c) rotations about the transverse axis.

3. The transverse axis is directed from right to left and passes through the center mass of the heart. Forward rotation about this axis results in some displacement of the top of the heart forward and the base of the heart backward; backward rotation results in the opposite changes (Fig. 15c).

In actual life simultaneous rotation of the heart around all these three axes is often met, and it is not by chance that Ashman describes 45 different positions of the heart in the thorax (according to Goldberger, 1954). In such cases, especially if there are simultaneous changes in the shape of the heart as a result of some heart disease, X-ray determination of the position of the heart is very difficult. Electrocardiography with the use of 12 leads for certain positions of the heart turns out to be of great help; the theory of Einthoven's triangle plays, and will continue to play, a definite role in this. /66

1. Einthoven's Theory

This theory (it may also be called a hypothesis) is based on the following suppositions:

1. Each separate moment of the stimulation process in the heart is like one dipole placed in the center of a triangle and which has the positive and negative poles very close together.

2. The apex of the triangle consists of the axillae and the left lower limb (R, L, F), which are located at an equal distance from the heart and from each other. In this way an equilateral triangle is formed, the sides of which are the three standard leads (we are not talking about the anatomical, but about the electrical, equilateral triangle.)

3. Since, with respect to the distance between the two poles of the dipole, the apex of the triangle is located at a very great distance from the heart, it may be considered that the triangle has a very large, almost infinite, area.

4. It is supposed that all tissues and organs located inside the limits of the triangle have the same resistance thanks to which a homogeneous medium is created.

5. The heart and the three limbs form one common surface which almost conforms to the frontal surface of the body.

Einthoven's theory is somewhat open to criticism since all five of the points described are quite conditional and approximate. However, in the practical sense, this theory turns out to be of great help and, as will be seen later, may be the basis for the development of various theoretical and practical problems of electrocardiography.

The dipole, which originates at each separate moment of the stimulation process, may be described as a vector having in correspondence with the characteristics of the electrical forces of a given moment, a definite amplitude and direction. This vector is called the electrical moment axis of the heart. During the entire process of depolarization of the ventricles there arise multiple electrical moment axes, the mean vector of which is called the mean electric axis of the heart, or more accurately, the axis of stimulation of the ventricles. In this way it is possible to represent both the mean electric axis of the process of recovery (repolarization) of the ventricles /67 and the stimulation of the auricles.

Let us suppose a normal electrical axis AB in the center of Einthoven's triangle (Fig. 16). The direction of the axis is defined by angle α , which is formed between the electric axis and the horizontal line, passing through the center of the triangle parallel to the axis of the first lead. The amount of the potential recorded by each lead, will depend on how the electric axis is inclined to the axis of the given lead (1). Thus, if the electric axis is parallel with respect to the axis of the lead, the given lead will record the maximum potential. With a gradual change in the

(1) The straight line which joins the points of placement of two electrodes of the lead is the axis of that lead.

direction of the electric axis from the direction of the axis of the lead, the amount of the registered potential gradually decreases; it reaches zero when the electric axis occupies a position perpendicular to the axis of the lead. This regularity is related to degree of inclination of the electric axis to the axis of the given lead (for the inclination, it is necessary to drop perpendiculars from the two ends of the electrical axis to the axis of the lead). As is seen in Fig. 16, it is characteristic for a normal position of the electric axis that the largest potential is recorded in the second lead ($a_2 b_2$), and the smallest in the third lead ($a_3 b_3$); in this respect the first lead occupies a middle position ($a_1 b_1$).

This is in accordance with Einthoven's law: $II = I + III$, i.e. the electromotive force, recorded from lead II, is equal to the algebraic sum of the electromotive forces of leads I and III. This law is proved by the following two methods:

1. Taking as a base that $II = I + III$, we may proceed retrospectively. It is known that $I = VL - VR$, $II = VF - VR$ and $III = VF - VL$; after substituting in the first formula we get: $VF - VR = (VL - VR) + (VF - VL)$, or $VF - VR = VF - VR$.

2. Using the fact that the leg is equal to the hypotenuse, multiplied by the cosine of the adjacent angle, it may be proved by means of mathematical calculation that:

$$\begin{aligned} a_1 b_1 &= e_1 = E \cos \alpha \\ a_2 b_2 &= e_2 = E \cos (\alpha - 60 \text{ deg.}) \\ a_3 b_3 &= e_3 = E \cos (120 \text{ deg.} - \alpha) \end{aligned} \tag{1}$$

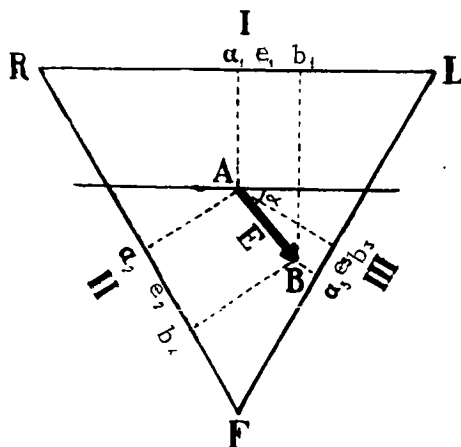


Fig. 16. Einthoven's triangle, the electric axis of the heart and Einthoven's law (explanation in text).

(E is the intensity of the electromotive force of the electric axis, and e is the intensity in the corresponding leads.) From this it follows that: /68

$$E \cos (120^\circ - \alpha) = E \cos (\alpha - 60^\circ) - E \cos \alpha, \quad (2)$$

thus,

$$e_3 = e_2 - e_1 \quad (3)$$

or

$$e_2 = e_1 + e_3 \quad (4)$$

consequently,

$$II = I + III. \quad (5)$$

In this way the amplitude of the waves of the electrocardiogram in the standard leads depends on the position of the heart in the thorax, or more accurately, on the position of the mean electric axis, i.e., on angle α . If the heart takes a horizontal position, the angle α decreases and the maximum electromotive force is recorded on lead I, and the minimum on lead III. The converse pattern is observed when the heart is in the vertical position. And so, by means of the standard leads, it is possible to determine the position of the heart in the thorax (see page 75). However, this method does not give complete data; as it takes into account only the rotation of the heart around the anteroposterior axis.

2. The Electric Positions of the Heart

It is known that when the heart is in the horizontal position, the left ventricle is turned toward the left shoulder and the right ventricle toward the left thigh; as a result, a complex of the type qR is recorded in the lead aVL, and in the lead aVF there appears a complex of the type rS. In the vertical position almost the opposite pattern is observed; in the lead aVL a complex of the type rS, appears and in the lead aVF, of the type qR. Proceeding purely from electrocardiographical considerations, and based on the similarity of shape of the complex QRS in leads VL and VF with the pattern in the right (V_1, V_2) and the left (V_5, V_6) pericardial leads, Wilson et al., (1944) describes six electric positions of the heart:

1. The horizontal electric position where there is similarity of the complexes QRS in the leads VL and $V_{5,6}$ and in the leads VF and $V_{1,2}$; the position of the electric axis is about -30° deg.

2. The semihorizontal electric position where in leads VL and $V_{5,6}$ there is a similarity of the QRS complexes, lead VF is distinguished by

the small amplitude of the complexes, and the position of the electric axis is about 0 deg.

3. The intermediate electric position where in leads VL, VF and V_{5,6} there is noted a similarity of the complexes QRS, the electric axis is located around +30 deg.

4. The semivertical electric position where in lead VF the QRS complexes resemble the complexes of leads V_{5,6} the lead VL is distinguished by a small amplitude, and the localization of the electric axis is about +60 deg.

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5. The vertical electric position where in lead VF the QRS complexes are similar to the complexes in leads V_{5,6}, and in lead VL, to the complexes in leads V_{1,2}; the position of the electric axis oscillates around +30 deg.

6. The indefinite electric position where no similarity of QRS complexes is noted in these leads.

These described electric positions do not always concur with the corresponding anatomic positions of the heart. In addition, account is taken only of rotation of the heart around its anteroposterior axis. However, the given method of determining the position of the heart has definite advantages over previous methods of determination using standard leads: it is based on the data of various leads (pericardial and limb leads) and is equally valuable in normal as well as in pathological conditions.

3. Determination and Classification of the Positions of the Heart According to Goldberger

An original and unusual method for the determination of the position of the heart by calculating its rotations around all three axes is proposed by Goldberger (1954). The author uses as a basis the five fundamental forms of the ventricular complex. By the appearance in one of the unipolar leads from the limbs of a definite form of ventricular complex, the conclusion may be drawn that the part of the heart corresponding to the given data is turned in the direction of the given lead. Based on this conclusion, he describes the following pattern characteristics of the rotations of the heart around its three anatomic axes.

A. Rotations of the heart around its anteroposterior axis. For the vertical position of the heart, it is characteristic to observe the QS wave or rS in lead aVL (the QS wave may be observed after anterior infarction, but with the heart positioned horizontally). In the horizontal position the qR wave or QR is observed in lead aVL.

B. Rotations of the heart around its longitudinal axis. During a mod-

erate clockwise rotation the rS wave appears in lead aVR; during considerable rotation the QR wave or Qr or qR appears. During moderate counterclockwise rotation in the pericardial leads, beginning with the position V₃ or V₄, there the qR wave appears; in the presence of considerable rotation these forms are observed beginning from lead V₂, and only in lead V₁ does the rS wave appear.

C. Rotations of the heart about its transverse axis. If the top of the heart is turned forward, the qR wave appears in lead aVF; if it rotates backward a complex of the type rS or RS appears in the given lead (such a complex may also be observed during the turning of the top forward, if the heart is horizontal).

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Based on this data, Goldberger, (1954), proposes the following classification of the various positions of the heart (Fig. 17).

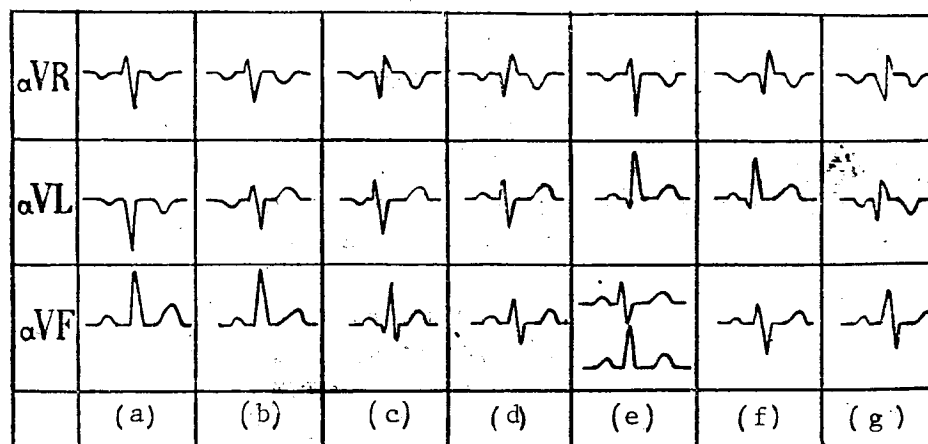


Fig. 17. Electrocardiogram from augmented unipolar leads during various positions of the heart according to Goldberger. The normal vertical heart with: (a) moderate counterclockwise rotation and rotation of the top forward, (b) moderate clockwise rotation and rotation of the top forward, (c) considerable clockwise rotation and rotation of the top forward and (d) considerable clockwise rotation and rotation of the top backward. The normal horizontal heart with: (e) moderate counterclockwise rotation and rotation of the top forward, (f) considerable clockwise rotation and rotation of the top forward and (g) considerable clockwise rotation and rotation of the top backward.

A. The normal vertical heart:

a) with a moderate counterclockwise rotation and rotation of the top forward (Fig. 17a);

b) with moderate clockwise rotation and rotation of the top forward (Fig. 17b);

c) with considerable clockwise rotation and rotation of the top forward (Fig. 17c);

d) with considerable clockwise rotation and rotation of the top backward (Fig. 17d).

B. The normal horizontal heart:

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a) with a moderate counterclockwise rotation and a rotation of the top forward (Fig. 17e);

b) with considerable counterclockwise rotation (rare);

c) with moderate clockwise rotation and rotation of the top forward (Fig. 17f);

d) with considerable clockwise rotation and rotation of the top backward (Fig. 17g).

Although the classification of Goldberger and his method of determining the various positions of the heart are comparatively more complete than other methods, nevertheless the individual positions may be disputed. Thus, Lenegre, Carouso and Chevalier (1954) find that Goldberger's method may be applied only in those cases, where there is no hypertrophy of the ventricles, or at least in those cases where there is only left ventricular hypertrophy or hypertrophy of both ventricles, but a normal relationship of the masses of the ventricles (coefficient left ventricle/right ventricle) remains without great changes. In those cases where this relationship changes (as, for example, right ventricular hypertrophy) the criteria of Goldberger lose their significance. In addition, as noted by M. B. Tartakovskiy, in right ventricular hypertrophy, blocks in the foot of the bundle of His and myocardial infarctions, it is very difficult, and sometimes even impossible, to work out a representation of the electric position of the heart according to the form of the QRS complex in the leads from the extremities, since the form of these complexes is considerably changed. Sodi-Pallares (1956) does not agree with Goldberger's opinion that the rS wave in lead aVR is explained by the turning of the right shoulder toward the cavity of the right ventricle. He asserts that the given lead is oriented toward the epicardial surface of the right auricle and the right ventricle. This same explanation is given by Sodi-Pallares (1956) for the genesis of the QS wave in the aVL lead; he relates it to the epicardial surface of the left auricle.

We find that the method of Goldberger, and, in general, his approach to the solution of these problems are of great interest and enrich the thinking of the specialist. In our practice we use this classification in a somewhat simplified form, but at the same time, for the determination of the mean electric axis of the heart we also use the stated theories of

Einthoven. While allotting great significance to the electrocardiographic method in determining the general position of the heart, we nevertheless find that this problem demands further clinical and experimental development, since it does not yet make definitely clear the relationship between the anatomic and electric positions of the heart.

VI. SOME ADDITIONAL QUANTITATIVE INDICES OF ELECTROCARDIOGRAMS

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1. Mean Electric Axis of the Heart ($\hat{A}QRS$)¹

As we have already stated, the mean electric axis of the heart is the mean vector of all moment vectors originating during the excitation of the ventricles (see page 66). In order to determine this index, a number of methods are used, among which we have the following.

A. Determination of the electric axis by means of the Einthoven triangle. (Fig. 18). In accordance with the polarity of the standard leads, each side of the triangle is divided into two equal halves, a positive side and a negative side. The center of each side connects with the opposite vertex of the triangle; the point of intersection of these lines determines the center of the triangle. This center is used, and a circle is drawn to enclose the three vertices of the triangle. The diameter of the circle, which is parallel to the side of lead I of the triangle, divides the circle into two semicircles. The position of the electric axis is determined by the magnitude of angle α which is formed between the axis and the designated diameter; the designation of the degrees is usually begun from the left side of the circle -- the upper semicircle (180°) is designated as negative and the lower semicircle as positive.

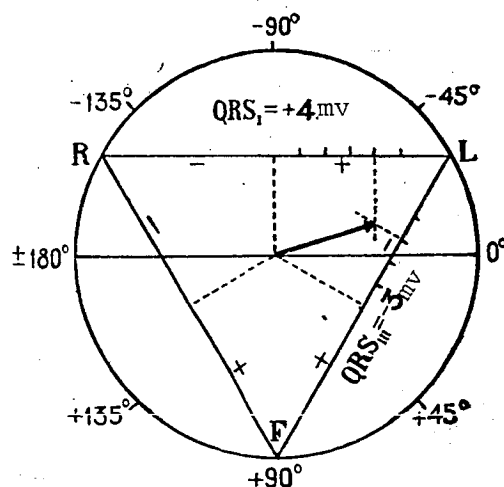


Fig. 18. Determination of the electric axis of the heart by means of Einthoven's triangle (explanation in the text).

¹ \hat{A} - the first letter of the word "axis"; the diacritical mark indicates the vectorial character of the phenomenon.

Then, the magnitude of the QRS complexes in leads I and III are determined (the algebraic sum of the amplitudes of the positive waves and negative waves). Let us allow that $QRS_I = +4$ mv, and $QRS_{III} = -3$ mv. The four conventional divisions are marked on the positive side of the axis of lead I and three of the same divisions are marked on the negative side of lead III. Perpendicular lines are dropped from the ends of these segments in leads I and III. The line which connects the point of intersection of these perpendicular lines to the center represents the electric axis; its position can be determined in degrees (angle α) and the magnitude can be determined in millivolts (according to conventional division).

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In practical work Einthoven's triangle method is not employed in the above manner, but rather according to special charts, for instance according to the Dieuaide chart (1921). With the aid of this chart the magnitude of angle α is easily determined.

B. Determination of the electric axis with the aid of multi-axial systems. A bipolar triaxial system is formed by standard leads. On plane RLF three lines corresponding to the axes of leads I, II and III cross at the common point (coinciding with the point of zero potential of the heart), forming 60° angles. The system is designated according to the polarity of the segments (Fig. 19a). In an analogous manner, they form a unipolar triaxial system using the axes of leads aVR, aVL, aVF and designating the corresponding polarity (Fig. 19b). By connecting the aforesaid triaxial systems it becomes possible to obtain a hexaxial system having six sextants (Fig. 19c); the angles between the sextants equal 30° .

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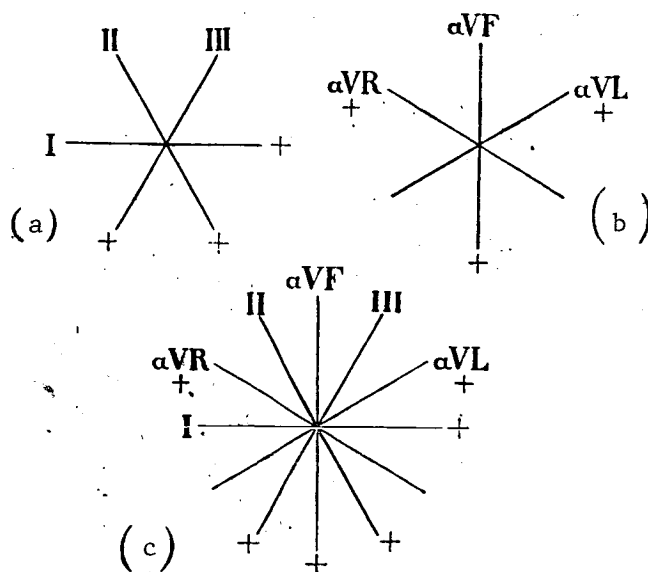


Fig. 19. Multi-axial systems: (a) bipolar triaxial system; (b) unipolar triaxial system; (c) hexaxial system.

By using the aforementioned multiaxial systems we can determine the electric axis in the same way as has been described to us above with reference to the triangle method. However, it should be noted that when using the triaxial system with standard leads we obtain an amplitude of the electric axis which is 1.15 times greater than when using the tri-axial system of unipolar limb leads (Goldberger, 1954).

In the determination of the electric axis by means of the above-described methods we used as a base the measurements of the amplitude of the QRS complex in millivolts; it is understood that in order to obtain precise data it is necessary to attach the proper leads at the same time. However, a number of authors find that the electric axis should not be determined by the amplitude of the QRS complex; for its determination we should base our computations on the dimensions of its surface. In this case, as considered by Sodi-Pallares (1956), we eliminate the inaccuracy which comes about due to the fact that the vertices of the corresponding waves do not appear at the same time in the various leads; if we take the points at the same time in the various leads then the mean electric axis is not measured but the moment axis is measured. The peak of the wave is determined by multiplying the amplitude (mm) by the base (sec); the figure which is obtained in this way is expressed in millivolt-seconds (one millivolt-second of the area = 1 mm X 0.01 sec.) or, in Ashman units, one Ashman unit is equal to four millivolt-seconds.

C. Determination of the electric axis by means of rectangular coordinates. Goldberger, in 1954, proposed to take the axes of leads I and aVF and to set up a system of coordinates (Fig. 20). By the usual method (as indicated above) we determine the direction and the magnitude of the electric axis (but amplitude QRS in lead aVF is multiplied by 1.15 and the magnitude which is obtained in this way is then transposed to the system of coordinates).

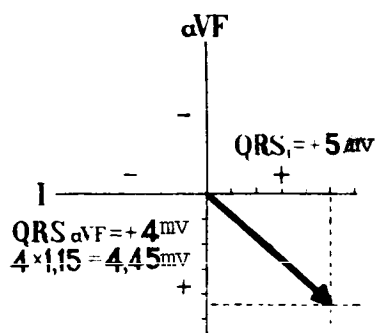


Fig. 20. The determination of the electric axis of the heart by means of rectangular coordinates (explanation in the text).

In all of the methods which have been described, the position of the electric axis is determined by the magnitude of angle α . It has been determined that the normal position of the heart has an electric axis of from $+20^\circ$ to $+70^\circ$. The deviation of the electric axis along the needle (greater than $+70^\circ$) should be indicated as a deviation to the right (clockwise) and its deviation counter to the needle (below $+20^\circ$) should be indicated as a deviation to the left (counterclockwise).

D. Determination of the electric axis by means of formulas. Already /75 in 1913 Lewis had proposed the following formula:

$$\text{Lewis index} = (R_I - R_{III}) + (S_{III} - S_I)$$

The amplitude of the waves is determined in millimeters; if the amplitude of the Q wave is greater than the amplitude of the S wave then the figures for the Q wave are used in the formula. In normal experiments the Lewis index fluctuates from $+20$ to -15 mm. It has been established that the index which drops below -15 mm indicates a clockwise deviation of the axis and that an index which goes above $+20$ mm indicates a counterclockwise deviation of the axis. Similar formulas have also been presented by other authors. White and Bock, in 1918, suggested setting up a formula $(U_I + D_{III}) - (D_I + U_{III})$, where U is the amplitude in meters of the greatest positive wave and D is the greatest negative wave in the corresponding standard leads. In 1947 Jinich, according to Sodi-Pallares, 1956, gave the index $(U_{aVL} + D_{aVF}) - (U_{aVF} + D_{aVL})$, where the same magnitudes are selected as in the preceding formula but in augmented unipolar limb leads; the normal magnitude of this index is ± 11 mm.

All of these formulas only offer an approximate representation of the position of the electric axis. A number of authors consider these formulas to be of value when it is a question of determining ventricular hypertrophy. We find that it is not necessary to use these formulas because an approximation of the position of the electric axis can also be obtained by a simple inspection of the electrocardiogram.

Significance of the determination of the electric axis. Einthoven, Fahr and Dewaart, 1913, had already shown that the position of the electric axis provides an approximation of the anatomical position of the heart; an electric axis of from $+70^\circ$ to $+90^\circ$ is characteristic for a vertical position of the heart and an electric axis of from $+20^\circ$ to 0° is characteristic for a horizontal position of the heart. A change in the position of the heart is principally associated with mechanical factors such as an elevated diaphragm, constitutional moments, etc.

However, a change in the electric axis can also be observed in the case of pathological conditions occurring without any noticeable deviation in the normal position of the heart. In this respect the determination of the electric axis is of great value when it is a question of diagnosis of ventricular hypertrophy, since a clockwise deviation (shift) indicates left ventricular hypertrophy. But it should be

kept in mind that when we are faced with a case of hypertrophy of the ventricles the changes in the electric axis are associated with shifts in

the coefficient: $\frac{\text{mass of the left ventricle}}{\text{mass of the right ventricle}}$. It is evident that even under 176

normal conditions the coefficient shifts within rather wide limits so that some changes can be observed in the electric axis without the presence of hypertrophy. In addition, when hypertrophy is present, the electric axis changes not only in the frontal plane of the Einthoven triangle but also in the other planes (Singler, 1946) and this is not taken into consideration in the determination of the electric axis. It is true that methods have been presented for determining the spatial electric axis but they are complicated ones and cannot be widely utilized in clinical practice.

We find that in establishing the position of the heart or the hypertrophy of the ventricles the determination of the electric axis cannot be of conclusive value. Rather frequently, in the presence of all of the electrocardiographic criteria of hypertrophy of one ventricle or the other, the electric axis shifts within normal limits or even shifts to the reverse side. The magnitude of the electric axis can only provide indirect or relative assistance and only then when it is compared with other criteria which are typical for such conditions.

2. Axonometry

According to this method which was developed primarily by Zarday in 1940, the electric axis is determined not only by the QRS complex but also by all of the five waves of the electrocardiogram. The electric axes which are obtained can be plotted on a chart and an axonogram can be obtained on which are measured a number of angles, in particular the angle between $\hat{A}QRS$ and $\hat{A}T$ (up to 60°) and between $\hat{A}R$ and $\hat{A}S$ (up to 130°).

A. V. Holtzmann (according to L. I. Vogelsson, 1957) plotted the following data on a normal axonogram:

$$\begin{aligned}\hat{A}P &= \text{from } +15^\circ \text{ to } +90^\circ \\ \hat{A}Q &= \text{from } -90^\circ \text{ to } +135^\circ \\ \hat{A}R &= \text{from } -15^\circ \text{ to } +90^\circ \\ \hat{A}S &= \text{from } -90^\circ \text{ to } +135^\circ \\ \hat{A}T &= \text{from } 0^\circ \text{ to } +90^\circ\end{aligned}$$

We concur with the opinion of L. I. Vogelsson (1957) that this method does not provide great advantages and can prove to be useful only in certain cases.

3. The Ventricular Gradient

In the case of a single muscle fiber, the depolarization and repolarization processes take place in the same direction and with the same sequence. In this way, a diphasic curve is obtained in which the algebraic sum of the 177 planes or the mean electric axes of the QRS complex and the T wave equal zero, i.e. $\hat{A}QRS + \hat{A}T = 0$.

However, the repolarization process in various parts of the ventricle does not follow a single sequence and differs with respect to its direction from the depolarization process with the ensuing result that there shows up between $\hat{A}QRS$ and $\hat{A}T$ a gradient (\hat{G}), i.e., $\hat{A}QRS = \hat{A}T = \hat{G}$. In other words, the plane of the QRST complex constitutes the repercussion of those electric phenomena which spring up because of local variations in the potential during the excitation process (Wilson and associates, 1934). Thus, the presence of the ventricular gradient is a necessary part of the normal human electrocardiogram. Its absence, or even changes in it, indicate the presence of pathological processes in the myocardium.

If we keep in mind that $\hat{A}T = \hat{G} - \hat{A}QRS$, we may conclude that the various changes in the T wave are either associated with shifts in the ventricular gradient or with changes in the QRS complex. Wilson and associates, 1934, referred to the changes in the T wave which are associated with the changes in the ventricular gradient as primary changes, since in these changes the depolarization process is changed but the repolarization process continues on normally. Consequently, in the case of primary changes in the T wave, it can be assumed that disease is present in the myocardium and that metabolic changes are involved (for instance in the case of myocardial ischemia). The changes in the T wave which are dependent upon the changes in the QRS complex were referred to by Wilson as secondary changes. In this case the depolarization process changes and, in accordance with these changes, the repolarization process changes once again but the ventricular gradient does not change. Thus, the secondary changes in the T wave do not stem from disease in the myocardium but originate as a result of changes in $\hat{A}QRS$ in the case of ventricular hypertrophy, extrasystoles and bundle branch blocks. Finally, the latter ones are manifestations of myocardial disease but the changes in the T wave which are detected in this case depend upon changes in $\hat{A}QRS$.

In clinical practice the ventricular gradient is determined by means of planimetric measurements of planes $\hat{A}QRS$ and $\hat{A}T$ in leads I and III. The plane which is thus obtained is expressed in Ashman units. Then, one of the following two methods is used:

1. We plot $\hat{A}QRS$ and $\hat{A}T$ on a triaxial system of standard leads according to the usual manner. On their bases a parallelogram is drawn, the diagonal line of which represents the magnitude and direction of the ventricular gradient (Fig. 21).
2. Based on the data compiled by Grant in 1957, Ye. L. Kilinskiy in 1959 assumed that in the absence of the ventricular gradient, $\hat{A}T$ will equal

$\hat{A}QRS$. A hypothetical T wave of this type (referred to by Gardberg and Rosen in 1957 as a "regression T wave") is designated as $\hat{A}T_1$ by Kilinskiy and in fact the recorded T wave was designated as $\hat{A}T_2$. Thus the characteristic of $\hat{A}T_2$ depends upon the interrelationship between $\hat{A}T_1$ and \hat{G} , in which case, according to the rules for adding the vectors, it can be considered that $\hat{A}T_2$ is a diagonal line of the parallelogram formed with the aid of $\hat{A}T_1$ and \hat{G} . We next transpose planes $\hat{A}T_2$ and $\hat{A}T_1$ ($\hat{A}T_1$ equals the size of $\hat{A}QRS$ but has a reverse position) onto a common triaxial system of standard leads; then placing $\hat{A}T_2$ in the base as a diagonal line (as one side) the parallelogram is drawn. That side of the parallelogram originating from the common points is represented by \hat{G} (Fig. 21b). The magnitude of the ventricular gradient in the standard, depending upon the position of the heart in the thorax, fluctuates considerably; on the average it equals 13 Ashman units (with fluctuations within the range of from 2.5 to 23 Ashman units). Its direction fluctuates between -17° and 86° . During the clockwise rotation of the heart around the longitudinal axis the gradient shifts between -5° and 86° , and during counterclockwise rotation it fluctuates from 17° to 70° (Lenegre, Carouso and Chevalier, 1954).

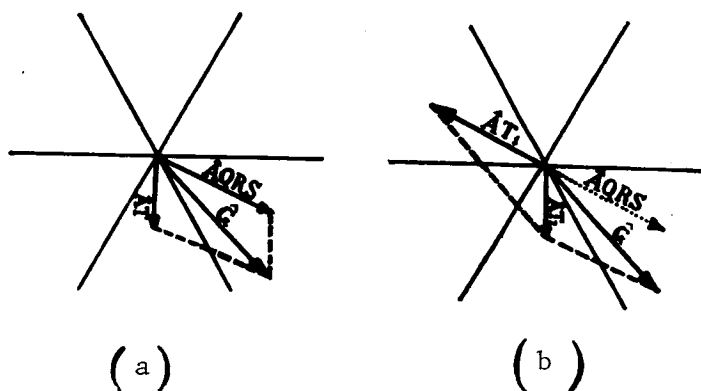


Fig. 21. Determination of the ventricular gradient: (a) first method; (b) second method (explanation given in the text).

In the technical literature a number of papers have been presented which deal with the theoretical and practical significance of the ventricular gradient (Ashman and Byer, 1943; Bailey, 1958, et al.). Our observations of hypertension (Table I) showed that, according to the degree of growth of the stages of the disease, the magnitude of the gradient decreases gradually, the QRS-T angle increases, and magnitude $\hat{A}T$ decreases. In addition to this, the sector of inclination gradually deviates to the left and angle $\hat{A}QRS-\hat{G}$ increases somewhat whereby the gradient often shifts clockwise of $\hat{A}QRS$. A detailed analysis of all of the criteria having to do with the ventricular gradient made it possible for us to determine the primary characteristic of of the changes in the T wave. It should be noted that the significance of

the ventricular gradient increases if there is simultaneously carried out a vectorcardiographic or a vectorelectrographic study. The determination of the ventricular gradient makes it also possible to study such criteria as the magnitude and position of $\hat{A}QRS$ and $\hat{A}T$, also the dimension of the QRS-T angle. These criteria are very useful in carrying out a vectorial analysis of the electric currents of the heart.

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Table I

Indices of the ventricular gradient, $\hat{A}T$ and $\hat{A}QRS$

COMPONENT	STAGES OF THE DISEASE		
	PRIMARY	SECONDARY	TERTIARY
Angle QRS-T (in degrees)	19.28 ± 12.39	57 ± 18.64	64.58 ± 23.47
Magnitude of $\hat{A}QRS$ (in Ashman units)	8.57 ± 3.33	8.28 ± 3.09	8.50 ± 3.32
Magnitude of $\hat{A}T$ (in Ashman units)	8 ± 3.75	6.87 ± 2.99	5.70 ± 3.00
Sector location of \hat{G} (in degrees)	$+38.57 \pm 16.17$	$+32.4 \pm 15.16$ (79%) -35 ± 18.37 (21%)	$+38.33 \pm 19.72$ (46%) -20 ± 18.93 (54%)
Magnitude of \hat{G} (in Ashman units)	18.21 ± 5.62	12.70 ± 6.37	11.3 ± 5.94

VII. NORMAL ELECTROCARDIOGRAM IN VARIOUS LEADS

Except for the P-Q segment, the configuration of all of the other components of a normal cardiogram differ greatly in the various leads. Segment P-Q has practically the same role in all of the leads. If the P-Q segment is of relatively equal length in the various leads, then it is necessary to measure the length of the P-Q segment in that lead where it is shortest.

1. Standard Leads

The P wave in leads I and II is always positive, in lead III it may be positive, diphasic or negative, and it can also be totally absent. According to the data of Ashman and Hull (1941), its amplitude in lead I does not exceed 1.1 mm., in lead II it does not exceed 2.5 mm. and in lead III, 2 mm. Its duration fluctuates between 0.06 and 0.10 sec., depending upon the frequency (rate) of cardiac activity.

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The QRS complex changes significantly at various positions of the heart.

The Q is often absent in the standard leads but it can also be observed in all of the three leads in approximately 10 per cent of the cases. It is more frequently detected in leads II and III; it is detected less frequently in lead I. According to the data by Kossmann and Johnston (1935), its maximum amplitude in lead I attains up to 1.5 mm. while attaining up to 2 mm. in leads II and III. Its width in these leads does not exceed 0.03 sec.

According to data compiled by the authors, the amplitude of the R wave fluctuates within the range of 1.5 to 19.4 mm. in lead I, from 4.0 to 22.0 mm. in lead II, and from 1.2 to 18.0 mm. in lead III. Its width fluctuates between 0.05 and 0.08 sec.

The S wave is encountered relatively more frequently than the Q wave—more so in leads I and II than in III, however its greatest amplitude is observed in lead III. According to L. I. Bogelson (1957), its amplitude in these leads fluctuates between 0 to 6 mm. The width of the S lead generally does not exceed 0.04 sec.

Thus, the amplitude of the entire QRS complex fluctuates within the range of from 5 to 20 mm. and its width fluctuates between 0.06 and 0.10 sec. In analyzing the width of the QRS complex it is necessary to take the recording in that lead where it is the smallest.

The described pattern of the QRS complex is observed in the normal position of the heart when the maximum amplitude of the waves appears in lead II and the minimum amplitude appears in lead III. In the case of a horizontal position of the heart the greatest amplitude is observed in lead I and the smallest amplitude is observed in lead III. A reverse pattern is encountered when the heart is in a vertical position. The amplitude of the waves in all of the standard leads decreases when the peak moves upwards since in this case $\hat{A}QRS$ assumes a more perpendicular position to the frontal plane of the Einthoven triangle. When the peak moves downwards the reverse is noted.

The RS-T segment is usually found at the level of the isoelectric line. In practice its upward deflection up to 1 mm. or its downward deflection down to 0.5 mm. should be taken as normal and if the T wave follows it there often is a somewhat upward deflection. In the presence of a negative T wave the segment deflects in a more downward direction. Its duration fluctuates between 0 to 0.15 sec.

The T wave often has the same direction as the greatest wave in the QRS complex. In leads I and II it is always positive and in lead III it is positive, diphasic or, relatively less frequently, negative. According to the data of Kossmann and Johnston (1935), the amplitude of the T wave in lead I fluctuates from 1 to 5.5 mm., in lead II it fluctuates from 1 to 6 mm. and in lead III from 0 to 3 mm. When the heart is in a vertical position a reverse pattern is observed.

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The U wave in the standard leads is encountered rather seldom and when it is presented it is usually small (0.15 mv according to Lepeschkin, 1951) and most readily observed in lead II.

2. Amplified Unipolar Limb Leads

In these leads we observe an unusual pattern in the normal electrocardiogram.

A. Lead aVR. The P wave is always negative.

The QRS complex is associated with types QS, Qr, rS, rSr', i.e., the negative wave always prevails here. However, sometimes complexes where the R wave prevails can also be observed, for example, QR or even qR. Goldberger (1954) believed that in such cases the peak moves backwards or the heart revolves clockwise about its longitudinal axis. Lenegre, Carouso and Chevalier (1954) found that in the case of a complex of this type particular care should be taken as regards the presence of a pathological condition in the heart; a complex of this type is often encountered in right ventricular hypertrophy.

Wave T is always negative with an amplitude of up to 6 mm.

B. Lead aVL. Wave P may be either positive or negative, the first form generally being observed when the heart is in a vertical position and the second form being observed when the heart is in a horizontal position. Diphasic P waves or even isoelectric P waves are encountered rather infrequently.

Complex QRS is different because of its great degree of variability and Davies was correct in 1958 in assuming that the interpretation of the variety of forms of QRS in lead aVL presents great difficulty. It can generally be considered that when the heart is in a vertical position complexes are recorded with a predominantly negative fluctuation (QS, rS, rSr') since the exploring electrode rotates in this case towards the cavity of the left and right ventricles and partially towards the epicardial plane of the right ventricle. In the case of a horizontal position of the heart, complexes are recorded having a predominance of positive fluctuation (qR, qRs, R, QR) since in this case the exploring electrode rotates basically towards the epicardial plane of the left ventricle. Nevertheless, Goldberger (1954) found that the amplitude of the R wave should not exceed 13 mm. He considered the criterion for a normal Q wave in this lead to be the following moments: its amplitude should be half of the amplitude of the R wave and the width should not exceed 0.04 sec.; the RS-T segment should be isoelectric and the negative P wave should be present. Many do not agree with the last of Goldberger's criteria, including M. B. Tartakovskiy (1958).

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Depending upon the position of the heart, the T wave is usually positive, less frequently isoelectric or negative, and its maximum amplitude does not exceed 4 mm.

C. Lead aVF. The P wave is positive in the majority of cases. In the case of a vertical position of the heart there may from time to time occur a rather small negative P wave.

The QRS complex refers to types QS, rS, RS, Rs, R, qR. When the heart is in a vertical position, complexes are observed having a predominance of positive fluctuation; for example qR, since in this case the exploring electrode generally moves towards the epicardial plane of the right ventricle. A normal R wave should not exceed an amplitude of 20 mm. A normal Q wave is characterized by the fact that its amplitude is less than half the amplitude of the R wave, the width is less than 0.04 sec.; the RS-T segment is isoelectric and the T wave is positive.

The T wave is usually positive with an amplitude up to 5 mm. In individual cases when the heart is in a horizontal position this wave can be absent or it can be negative with a very small amplitude. Generally speaking there is a definite relationship between the shapes of the T wave (and also of the Q wave) in leads aVF and III.

Table 2

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Normal magnitudes of the waves of the electrocardiograms in amplified unipolar limb leads according to M. B. Tartakovskiy, 1958

LEAD	ELECTRICAL POSITION OF THE HEART	MAGNI- TUDE	WAVE				
			P	Q	R	S	T
aVR	Vertical and semivertical	mean	-1.1	8.4	1.1	11.1	-3.3
		max.	-3.0	15.0	2.5	17.0	-7.0
		min.	-0.5	- ¹⁾	-	-	-0.5
	Intermediate	mean	-1.2	8.8	1.0	10.5	-3.2
		max.	-2.5	14.0	2.5	16.0	-7.5
		min.	-0.5	-	-	-	0.5
	Horizontal and semihorizontal	mean	-1.1	9.0	1.4	12.8	-3.0
		max.	-3.0	18.0	4.0	15.0	-5.0
		min.	-0.5	-	-	-	-1.0

¹The minus (-) sign indicates the absence of the wave in question.

Table 2 (Continued)

Normal magnitudes of the waves of the electrocardiograms in amplified unipolar limb leads according to M. B. Tartakovskiy, 1958

LEAD	ELECTRICAL POSITION OF THE HEART	MAGNITUDE	WAVE				
			P	Q	R	S	T
aVL	Vertical and semivertical	mean	0.2	1.2	3.1	3.9	0.8
		max.	1.5	2.5	8.0	10.0	4.0
		min.	-0.5	-	1.0	-	-3.0 ¹⁾
	Intermediate	mean	0.6	0.8	6.4	1.8	2.1
		max.	2.0	2.0	15.0	3.0	6.5
		min.	0	-	4.0	-	0.5
	Horizontal and semihorizontal	mean	0.5	1.1	9.5	1.6	2.1
		max.	1.5	3.0	17.0	3.0	6.0
		min.	-0.5	-	4.5	-	0.5
aVF	Vertical and semivertical	mean	1.0	1.0	10.4	1.7	2.5
		max.	3.0	6.5	23.0	3.0	6.0
		min.	-0.5	-	4.0	-	0.5
	Intermediate	mean	1.1	0.7	6.9	1.5	2.0
		max.	2.5	1.5	14.0	3.0	5.0
		min.	0	-	5.0	-	0
	Horizontal and semihorizontal	mean	0.9	1.1	2.9	3.2	0.8
		max.	2.5	2.5	8.0	12.0	3.0
		min.	0	-	1.0	1.0	-3.0

Goldberger (1954) has given a detailed description of the shapes of complexes at various classifications of heart positions (see page 69 of original text). M. B. Tartakovskiy (1958) has made clear the characteristic individual component features of the electrocardiogram in augmented unipolar limb leads in various electric positions of the heart according to Wilson (Table 2).

3. Precordial Leads

The P wave is generally positive and has a small amplitude. In lead V₁ and also in V₂ this wave can be diphasic or negative. The amplitude of the P wave increases somewhat depending upon the degree of proximity to the

¹The T wave is negative in the case of a ventricular complex of type rS or QS.

left positions.

In the case of a normal position of the heart, the QRS complex presents the following pattern: A complex of the rS type, or, infrequently, QS, is recorded in lead V₁. In lead V₂ the amplitude of the R wave decreases slightly or remains the same as that in lead V₁; complexes of the QS type are hardly ever encountered in lead V₂. In lead V₃ the R wave increases somewhat and the S wave decreases and the complex often assumes the RS shape. In lead V₄ the maximum amplitude of the R wave is recorded. The S wave decreases greatly and a small Q (qRs) wave appears. In leads V₅ and V₆ the amplitude of the R wave decreases slightly and the S wave often disappears. The Q wave remains, but in lead V₆ its amplitude is relatively decreased. Consequently, from the right to the left positions the amplitude of the S wave gradually decreases, the R wave gradually increases (the R/S coefficient decreases accordingly) and its maximum amplitude is noted in lead V₄. Starting from the latter position a small Q wave appears. Complexes of the intermediate type (RS) are recorded principally in segment V₃. Such a pattern can be explained by the fact that the right chest positions are recorded by the potential of the epicardial plane of the right ventricle which is characterized by the complexes of the rS type. The left positions deflect towards the epicardial plane of the left ventricle, and the electromotive force of the left ventricle brings about complexes of the qR type.

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At various positions of the heart the configuration of the QRS complex changes and the essentially predominating feature of the precordial leads is the shifting of the heart about its longitudinal axis. Thus, in the case of a shift about the given axis along the needle, the right ventricle occupies a large section of the heart's front surface, the result of this being that complexes of the rS type are also recorded in the intermediate zone and sometimes even further, e.g., in the left chest positions. A counterclockwise shift brings about a reverse pattern: the major portion of the front surface of the heart is occupied by the left ventricle and complexes of the qR type appear in the transitional zone and even in the right chest positions.

The RS-T segment, particularly in the right positions, is usually isoelectric with upward deflection up to 2 mm.; its downward deflection down to 0.5 mm. can also be observed in the standard.

The T wave is usually positive. In lead V₁ it may be negative, especially when the heart is in a horizontal position. Sometimes the negative T wave is also observed in lead V₂. But if the T wave is positive in lead V₁ then it cannot be negative in the subsequent positions. The amplitude of the T wave increases gradually from lead V₁ to lead V₄ and its maximum amplitude is noticed in lead V₄; in leads V₅ and V₆ the T wave

decreases somewhat. A definite association between the R waves and the T waves is noticed and from this point of view it is recommended that the R/T coefficient be determined.

Table 3 gives the data compiled by Sokolow and Lyon in 1949 concerning the normal indices of electrocardiograms in precordial leads.

The U wave is small and, according to Lepeschkin (1951), it attains a magnitude of 0.2 mv and is manifested less often in lead V_4 . Zuravitz, Kemp, and Bellet (1957) found that in the majority of cases the U wave has the same polarity as the T wave and their ratio is the same in all of the leads.

(Our norms of basic electrocardiographic indices are given on pages 325 and 326, orig. text).

Table 3

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Normal Indices of the ventricular complex of an electrocardiogram in precordial leads according to Sokolow and Lyon (1949)

INDEX			LEAD					
			V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Magnitude of the wave in mm.	Q	Mean	0	0	0.01	0.1	0.3	0.4
		Max.	0	0	0.5	3	3.0	2.0
		Min.	0	0	0	0	0	0
	R	Mean	2.3	5.9	8.9	14.2	12.1	9.2
		Max.	7.0	16.0	26.0	27.0	26.0	22.0
		Min.	0	0	1.5	4.0	4.0	4.0
	S	Mean	8.6	12.7	8.8	5.2	1.5	0.6
		Max.	25.0	29.0	25.0	20.0	6.0	7.0
		Min.	2.0	0	0	0	0	0
	T	Mean	0.15	5.2	5.38	4.8	3.43	2.43
		Max.	4.0	18.0	16.0	17.0	9.0	5.0
		Min.	-4.0	-3.0	-2.0	0	0	-0.5
R/S coefficient		Mean	0.3	0.2	1.4	4.1	7.3	9.0
		Max.	1.0	1.3	10.0	19.0	24.0	22.0
		Min.	0	0.1	0.1	0.2	1.0	2.0
R/T coefficient		Mean	1.4	1.4	1.9	2.9	3.5	4.1
		Max.	7.0	12.0	13.0	9.0	9.0	10.0
		Min.	0.3	0.2	0.3	0.3	1.0	1.7
Formation time of the internal deviations in sec.		Mean	0.02	0.025	0.03	0.034	0.04	0.04
		Max.	0.03	0.04	0.04	0.05	0.05	0.05
		Min.	0	0	0.02	0.02	0.02	0.02

VIII. PATHOLOGICAL CHANGES IN ELECTROCARDIOGRAMS

It is often quite difficult to distinguish the pathological pattern from the normal pattern, even though, as noted by Simonson (1958), the pathological changes can be studied more easily than diverse variants of the normal pattern. On a pathological electrocardiogram we can observe changes in amplitude, width and shape of the waves, displacements of the segments or changes in their length and shape, and changes in the normal sequence of the waves or complexes.

Further on we give, in a few words, the basic pathological changes of the individual components of an electrocardiogram.

1. The Auricular Complex

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An increase or a decrease in the amplitude of a P wave can be observed under pathological conditions. Sometimes the wave is level, i.e. it becomes isoelectric. In certain cases gigantic P waves develop with an amplitude of up to 10 mm., in particular they appear in lead V_1 . Changes in the

width of the P wave are noted when its duration increases by more than 0.1 sec. The changes in the shape of the P wave result in the appearance of expansions or notches on the rising or descending curves of the wave, in the formation of a double peak, flatness or tapering off of the peak, changes in the symmetry of the wave and in the appearance of diphasic waves (with a different sequence) or negative waves (positive in the aVR segment).

As is well known, the normal form of the P wave is chiefly explained by the fact that, in the first place, the normal impulse develops from the sinus node and, in the second place, it spreads into the auricular musculature radially in all directions. In such a case the P wave is referred to as a sinus wave, and it becomes positive in leads I, II and aVF, and negative in the aVR lead.

If the initial point of the impulses is situated in the right auricle, but in the vicinity of the sinus node, then the shape and the magnitude of the P wave change somewhat. However, if the initial point of the impulses is situated in the lower portions of the right auricle (or in the left auricle) then the polarity of the P wave changes; in leads II and aVF it becomes negative. With the development of the impulses from the atrioventricular node there is obtained a so-called nodal P which is characterized by a reverse polarity with respect to the sinus wave: it is negative in leads II, III, aVF, and positive in leads aVR and aVL. Such a pattern is explained by the fact that the excitation of the auricle takes place retrogradely from the side of the atrioventricular node. It should be emphasized that if the impulse originates in the peak of the node, then nodal P precedes the QRS complex, but there is noted somewhat of a contraction in the P-Q segment. If it originates in the middle part of the node then the P wave does not appear, because during the time of the synchronous excitation of the auricle and the ventricles it joins with the large ventricular complex. If the impulse originates in the lower portion of the node then nodal

P appears in the middle of the ventricular complex in the area of the RS-T segment, resulting in a relatively retarded excitation of the auricle.

Changes in the amplitude, width and shape of the P wave are associated principally with disease of the myocardium of the auricle. The extracardial nerves only play a relative role here because in this case it is not a question of the impulses passing through the special fiber but along the regular muscular fibers.

It is difficult to explain why in certain pathological cases the amplitude of the P wave increases. We can only tentatively agree with the opinion of L. I. Vogelsson (1957) that the increase in the electromotive force of the right auricle results in its decrease. It should be noted that the augmented and double-peaked P waves in leads I and II meet, in practice, in the presence of mitral stenosis and are designated as mitral P's, and the augmented and pointed P's in leads II and III, which appear basically during chronic ailments of the small cycle of blood circulation, are designated as pulmonary P's. /87

2. The P-Q Segment

The pathology of the P-Q segment consists principally in the changes in its duration and also in its deviation. The augmented segment is observed during the retardation of the atrioventricular conductance as a result of organic affections of these pathways or the impairment of the functions of the extracardial nerves, for instance, in the case of an increase in the tone of the vagus nerve. In individual cases, in particular in the presence of the Wolff, Parkinson and White syndrome (1930), there is observed an acute contraction and even the disappearance of the P-Q segment.

The deviation in the P-Q segment is noted principally during a deviation in the P-Ta segment. The deviation can be observed during auricular infarctions.

3. The Ventricular Complex

The pathological changes in the Q projection consist of the increase in its amplitude and width, with the formation on one of its nodes of expansions or notches. Quite typical in pathology is the appearance of a Q wave which was not previously there. Great importance is attached to the changes in the Q wave when myocardial infarctions are detected. However, it must be kept in mind that similar but reverse changes can also be observed in the Q wave in the case of so-called metabolic injuries in the myocardium (Goldman, Gross and Rubin, 1960).

The changes in the amplitude of the R wave consist in its increase, or in its decrease to the point of disappearing. The width of the R wave often increases, its shape changes as a result of the appearance of expansions or notches on its rising or falling nodes, segmentation of the peak of the wave (M-shaped R), contraction of the falling node with merging with the RS-T segment. Despite the opinion of some authors, we find that the R

1

wave cannot remain negative (just as the Q wave and the S wave cannot be positive). In the absence of an R wave, the observed complex is designated as QS (a V-shaped complex, according to Burch and Winsor, 1955), since without the presence of R it is difficult to determine whether the negative wave is a Q wave or an S wave.

The pathological changes in the S wave consist of the increase of its amplitude and width, the formation of expansions or notches on one of its nodes, the segmentation of the peak of the wave (W-shaped S) and also the contraction of the rising node.

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In this way, if we take the QRS complex as a whole, we may state that in pathology there can be observed an increase or a decrease in QRS's voltage, an increase in its width and changes in its configuration. These changes take place as a result of the impairment in the excitatory distribution process to the ventricles. This impairment, in turn, can be a result of the changed tone of the extracardial nerves, or, more frequently, a result of the development of muscular hypertrophic processes or the presence of diffused or focal changes in the myocardium.

The duration of the RS-T segment is principally associated with the frequency of the rhythm of the cardiac contractions; more precisely with the duration of the systole, whereby in the case of a short systole there is observed a decrease in the segment down to the appearance of an RS-T junction. The RS-T segment can deviate upwards (positive deviation) or downwards (negative deviation) from the isoelectric lines. If in one or several leads there is observed a deviation of the segment in the same direction, it is known as a concordant deviation; the reverse pattern is called a discordant deviation. The shape of the segment also changes frequently: a plateau-shaped, cupola-shaped, semilunar-shaped segment or other shapes are observed. Changes in the RS-T segment play an important role in the development of diffused changes, hypoxia myocardial infarctions and ventricular hypertrophies.

Changes in the amplitude of the T wave result in its increase or decrease down to planing (flattening out). The width of the wave can change both in the side of the increase and in the side of the decrease. Changes in the form of the T wave result in the appearance of symmetrical waves or even result in a change in the polarity and shape of the diphasic waves (with a different sequence of the phases) or of the negative waves (positive in lead aVR). Augmented, tapered and symmetrical T waves are sometimes characterized as gigantic, whereas deep, symmetrical, negative waves are characterized as coronary waves. The changes in the T wave are of great significance with respect to ascertaining the diffused and focal changes in the myocardium, hypoxia of the muscles of the ventricles, and their overexertion and hypertrophy. It should also be kept in mind that analogous changes can also be observed in healthy individuals during physical strain, intake of food, from the determined position of the heart (Gardberg and Rosen, 1957) or even from psychic experiences (Grau, 1956).

If the Q-T interval is considered as a whole, it may be stated that

in pathological cases its duration and configuration change. An extension of the electric systole can be observed under the most varied situations. A number of authors (Teren and Tsigali, 1947; Kornel and Braun, 1956) consider this to be typical in the case of active rheumocarditis. It is interesting to point out that Jervell and Lange-Nielsen (1957) reported a syndrome in deaf-mute children where in the absence of organic pathology in the heart there were observed a prolonged Q-T interval and periodic attacks of cardiac interruptions. Changes in the configuration of the QRST complex are principally associated with disturbances in the passage of the impulses in the ventricles, for instance in the case of intraventricular blocks, ventricular extrasystoles, hypertrophies, diffused and focal changes in the myocardium of ventricles, etc. In all of these cases when there is a disruption of the normal relative synchronism of excitation of the two ventricles and one of them is excited before the other, or if the excitation of the second ventricle is delayed, then characteristic changes are detected in the configuration of the ventricular complex combined with the appearance of a partial electric pattern prior to the excited ventricle. Complexes of these types can be referred to as ventricular complexes, in contrast to the usual supraventricular types which are formed during the normal mechanism of distribution of the excitation process. If among the usual complexes which move consecutively we observe such complexes whose shape differs somewhat from the shape of the other complexes, then it is customary to refer to these complexes as aberrant.

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In pathological cases the T wave can become negative (Surawicz, Kemp and Bellet, 1957) or the shape of the normal T-U junction can change (Holzmann, 1957). Fubretta and associates (1957) referred to the simultaneous changes in the U wave and in the T-U segment as a papillary syndrome. Ye.A. Aleksandrova and Ye.L. Kilinskiy (1961) gave a certain significance to the deviation of the T-U segment in pathological cases.

IX. ELECTROCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE SYNTHETIC ELECTROCARDIOLOGIC COMPLEX

1. Position of the Heart

The determination of the position of the heart in the chest has a diagnostic significance, just as the anatomic position of the heart and the corresponding electric pattern explained by it can be subjected to changes in the case of physiological conditions or numerous intracardiac and extracardiac conditions. In this aspect the determination of the position of the heart assumes an important role in differential diagnosis. It should be remembered that in the case of dextrocardia the electrocardiogram in lead I assumes a reflected image of the usual pattern for the given lead. Lead II suggests the pattern in lead III of a typical electrocardiogram and vice-versa. Similar relationships are also observed between the aVR and aVL leads. In this case the precordial leads should be recorded in the following sequence: V₂, V₁, V₃R, V₄R, V₅R, V₆R. A knowledge of these moments is of great importance, especially in the case of simultaneous presence of patho-

/90

logy in the heart (Fig. 22).

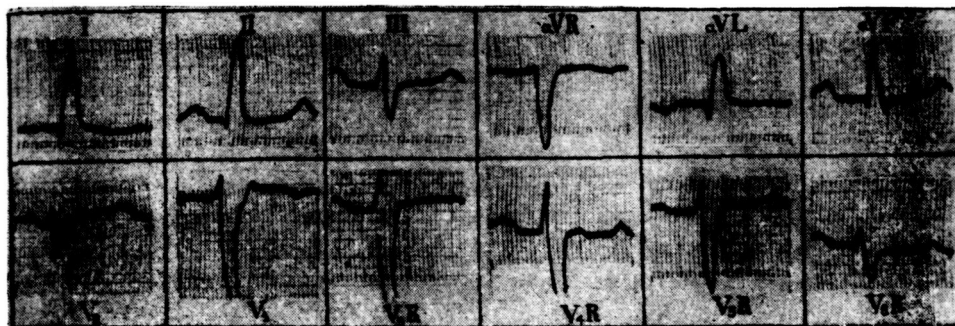


Fig. 22: Dextrocardia. Electrocardiogram of a patient, 14 years old, with the following diagnosis: tetralogy of Fallot. Right ventricular hypertrophy: the chest leads are recorded from left to right, lead I has a reflected image, lead II has the shape of lead III and vice-versa; there is also a relationship of this type between leads aVR and aVL.

The electrocardiograph is very useful in determining the position of the heart. We have already discussed in previous sections of this chapter the various methods of determining and classifying the positions of the heart and therefore we shall not dwell any further on this.

2. Impairment of the Automatic Process

The normal rhythm of the heart is of sinus origin and fluctuates in an adult heart within a range of 60 to 90 beats per minute. The impairment of the operation of the automatic process takes place in two ways. First, only the quantity can change and the sequence of the formation of impulses; the sinus node remains once again at its original point. These rhythms are designated as sinus or nomotopic rhythms. Secondly, the initial point of the impulses can change and the work of the heart is assured as a result of the operation of other automatic centers, i.e. the heart acquires a new pace-maker of its rhythm. Rhythms of this type are referred to as heterotopic rhythms.

A. Sinus rhythms

a. Sinus tachycardia. In this case the frequency of the cardiac contractions is greater than 90 beats in one minute, generally 110 to 120 beats. Sinus tachycardia is encountered quite frequently in various physiological as well as pathological conditions whereby great importance is placed on the tone of the extracardial nerves (the depressed tone of the vagus nerve or the increased tone of the sympathetic nerves result in a sinus rhythm), and also on such factors as insufficient supply of oxygen to the sinus node, the effect of toxic factors on the sinus or an increase in the temperature of the blood.

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The form of the auricular and ventricular complexes usually does not

change on the electrocardiogram, although acute tachycardia may cause certain changes. A contraction of the T-P segment develops as a result of a contraction in the diastole. In the case of a highly advanced degree of tachycardia, the P wave assumes a rounded shape and the T-P segment contracts somewhat; the P wave joins with the T wave of the foregoing complex, and certain difficulties in differential diagnosis thereby arise. In a case of rather frequent rhythm, we note a relative prolongation of the Q-T interval, a somewhat downward deviation of the RS-T segment, a change in the shape of the Q-T interval, a decrease in the amplitude of the T wave, and even the appearance of negative T waves.

b. Sinus bradycardia. In this case, the frequency of the cardiac contractions is less than 60 beats per minute - usually between 50 and 55 beats. This occurs under the most varied physiological and pathological conditions. In the formation of sinus bradycardia, great importance is attached to such factors as an increase in the tone of the vagus nerve, a decrease in the tone of the sympathetic nerves, the effect of toxic factors on the sinus node, and also age, the type of physique, and so forth.

The form of the auricular and ventricular complexes usually does not change on the electrocardiogram. A prolongation of the T-P segment and often some degree of prolongation of the P-Q segment are observed. Gross (1956) found that in the case of acute bradycardia sharp and notched P waves also appeared.

c. Sinus arrhythmia. In this case, an erratic alternation of the sinus impulses is observed. This arrhythmia is encountered in two forms. It is a characteristic feature of the respiratory form that the erratic alternation of the impulses is cyclical and in the inspirational phase the number of impulses increases and in the expirational phase the number of impulses decreases. This form is usually encountered in children, seldom in adults, and is associated with the refractory effect of the inspirational phase on the tone of the vagus nerve. The predominant effect of the vagus nerve is also corroborated by the fact that a sinus arrhythmia often accompanies a sinus bradycardia. This form of sinus arrhythmia is regarded as a physiological phenomenon, although, as indicated by Myers (1956), it can be an expression of hypersensitivity of the carotid area and can also be encountered in the case of heart diseases. The non-respiratory form of sinus arrhythmia is observed without dependence upon the phase of the respiratory cycle and is encountered, in particular, during various heart diseases which disrupt the nutrition of the myocardium or which result in organic disease in it. /92

On the electrocardiogram in Figure 23a, the shape of the auricular and ventricular complexes is generally not changed, but there is a periodic contraction and lengthening of the pause between the individual cycles of the heart (R-R intervals). It should be kept in mind that even without the presence of sinus arrhythmia these intervals are not sharply defined because a definite physiological mobility is characteristic for the functioning of a normal sinus node. It is customary to consider that if the difference between these pauses exceeds 0.15 sec., we may speak of a sinus arrhythmia.

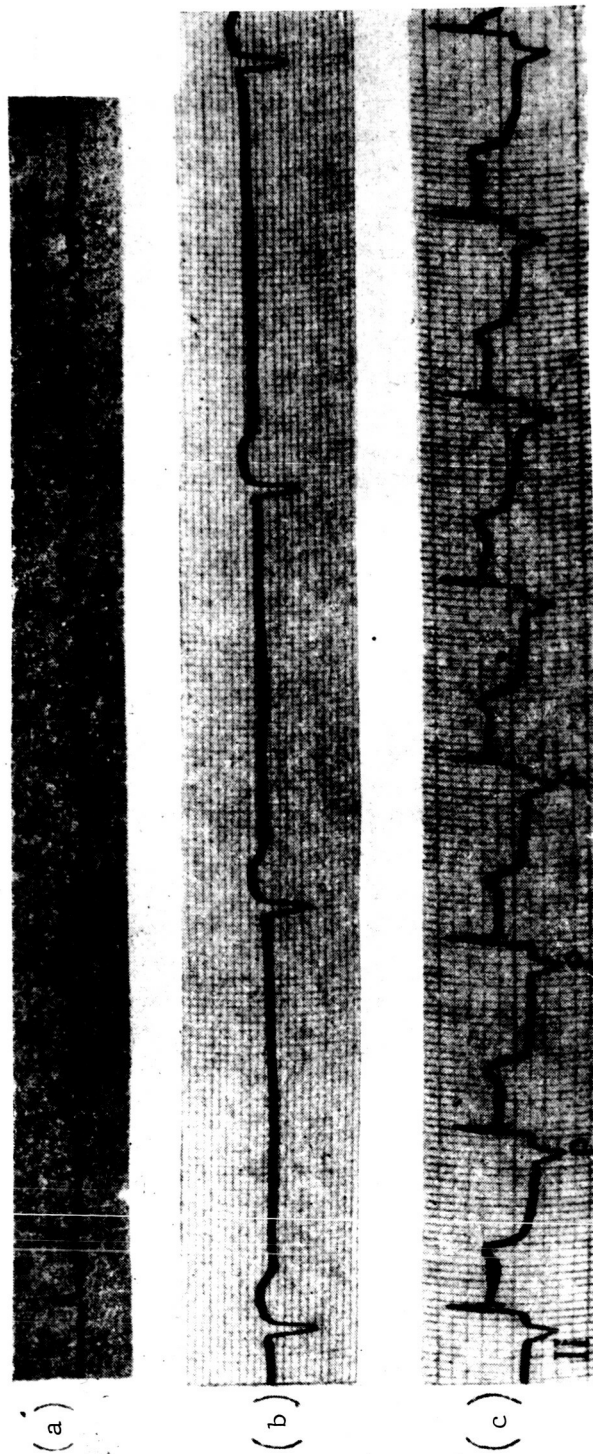


Fig. 23: Impairment of the automatic process. (a) Electrocardiogram of a 13-year-old child, I. A., who is, practically speaking, healthy. Sinus arrhythmia with migration of the cardiac pacemaker. (b) Electrocardiogram of a dog during artificial blood circulation. The nodal rhythm from the middle part has a frequency of 53 contractions per minute. The test was accompanied by a migration of the cardiac pacemaker, the appearance of ventricular rhythm, then there occurred a scintillation of the ventricles with ensuing standstill of the heart. (c) Electrocardiogram of a patient, P. E., 21 years old, with the following diagnosis: mitral defect with prevalence of stenosis. Nodal rhythm from the upper part with a frequency of 60 contractions per minute.

Katz and Pick (1956) differentiated between two types of arrhythmia. One type is encountered when the impulse constantly arises from the same point within the sinus node and the P wave and the P-Q segment have the same configuration in all of the complexes. The second type is encountered when there takes place a migration of the cardiac pacemaker within the sinus node as a result of which the form of the P wave and the length of the P-Q segment periodically change. We distinguish still another type when sinus arrhythmia accompanies the regular migration of the cardiac pacemaker (Fig. 23a).

B. Heterotopic rhythms

a. Nodal rhythm. As a result of the non-functioning of the sinus node the impulses arise from the atrioventricular node and spread into the ventricles along the usual pathways and retrogressively to the auricles. Consequently, the rhythm of the ventricular contractions slows down and averages from 35 to 60 impulses, and the lower the initial point of the formation of the impulses in the atrioventricular node, the slower the heart rhythm.

The nodal rhythm appears under all of these circumstances when the function of the sinus node is suppressed: digitalis toxicity, various diseases of the myocardium, sinoauricular block, and so on.

The electrocardiogram shows bradycardia, the form of the ventricular complex is usually unchanged, except that the auricular wave assumes a nodal character. There is principally observed a nodal rhythm coming from the upper part of the atrioventricular node. In this case, the nodal P wave is situated as far as the ventricular complex and the P-Q segment is relatively contracted (Figs. 23c, 24a). In a nodal rhythm coming from the middle part of the node, the P wave node is absent (Figs. 23b, 24b), and in the case of an infrequently observed rhythm from the lower part of the node, the P wave node is situated between the QRS complex and the T wave (Fig. 26b) (see page 86 of the original text for explanation). We concur with the opinion of Katz and Pick (1956) that such a differentiation of the types of nodal rhythms is very difficult. For example, in the case of a middle nodal rhythm, as a result of retarded retrograde excitation of the auricle, an electrocardiographic pattern of lower nodal rhythm can be observed. Another example is in the case of a slowing down of the conductance in the ventricles, as a result of the relatively retarded excitation of the ventricles in the low nodal rhythm. A pattern of a middle nodal rhythm or even of an upper nodal rhythm is thus observed. For this reason, these authors consider it more expedient not to define more accurately the point of origin of the impulses within the atrioventricular node and to designate all of the described types as nodal rhythms with retrograde conductance. /94

There is also distinguished a so-called rhythm of the coronal or coronary nodal rhythm. With this form, the impulses arise from the splitting of the upper part of the atrioventricular node in the area where the sinus is located. Nodal P wave is positive in lead I and is sometimes also positive in lead II.

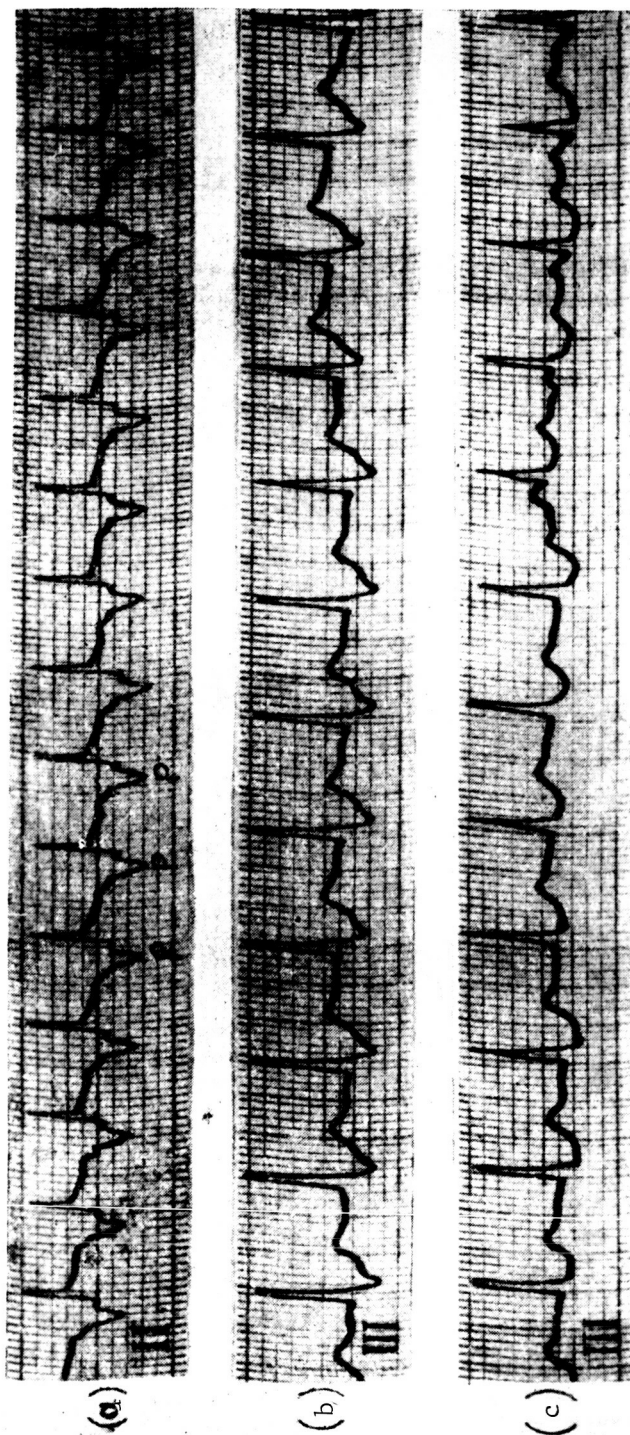


Fig. 24: Impairment of the automatic process. (a) Electrocardiogram of a patient, P. E., 21 years old, with the following diagnosis: mitral defect with prevalence of stenosis. The nodal tachycardia from the upper part has a frequency of 160 contractions per minute. (b) Electrocardiogram of another patient, B. N., 31 years old, with the following diagnosis: mitral defect with prevalence of stenosis and stenosis of the aorta. Nodal tachycardia from the middle part; (c) Electrocardiogram of the same patient. Interference with dissociation is noted following an aortic commissurotomy.

A nodal rhythm with an impulse frequency up to 100 beats and more per minute is encountered rather infrequently. Such a pattern occurs during nodal tachycardia as a result of increased irritability of the node. This type of nodal rhythm is referred to by Myers (1956) as active and the rest of the types he presents which arise as a result of depression of the function of the sinus node are referred to by him as passive. We often observed nodal tachycardia following heart surgery (Fig. 24a, b).

Ya. G. Etinger and V. Ye. Nezhlin (1932) proposed to include all of the above-mentioned types of nodal rhythms during which the impulses reach both the ventricles and the auricles in the nodal rhythm of the first type, in contrast to the nodal rhythm of the second type where the impulses from the atrioventricular node do not reach the auricles because the ventricles are also excited by the nodal impulse and the auricles by the sinus rhythm. Thus, in the second type, a dissociation of the auricles and the ventricles occurs (dissociation is the result of the interaction of two pacemakers of rhythm acting simultaneously). However, it is characteristic for the atrioventricular center to affect the impulses relatively more frequently than the sinus center. In this case, the movement of the sinus impulses along the normal pathways is not disturbed and the sinus impulse excites the ventricles each time that it forces them outside of the refractory phase. In this way, an interference of the sinus and nodal rhythms is obtained (the interference is a physiological result of the interaction of two rhythm pacemakers acting simultaneously or two different impulses arising immediately one after another). This means that the nodal rhythm of the second type should otherwise be referred to as a dissociation with interference (V. Ye. Nezhlin and S. Ye. Karpay, 1959). This is encountered in rheumatism, infectious diseases and digitalis intoxication. This has sometimes been observed by us in heart operations (Fig. 24c). G. G. Hellstein (1960) differentiates four types of dissociation. The first type is a sinus rhythm-dissociation-sinus rhythm; the second type is a sinus rhythm-dissociation-atrioventricular rhythm; the third type is atrioventricular rhythm-dissociation-atrioventricular rhythm; the fourth type is atrioventricular rhythm-dissociation-sinus rhythm. We believe that a classification of this kind has definite significance in the determination of the electrocardiographic changes during heart surgery. /96

b. Migration of the cardiac pacemaker. In this case one pacemaker acts for the entire heart, but it migrates periodically from the sinus node to the atrioventricular node and vice versa. Such deviations to and fro are also possible within the sinus node itself. This phenomenon can be observed in a completely healthy subject and also in persons affected with rheumatism, digitalis intoxication, and so forth. We consider that in all of these cases there occurs either an increase in the tone of the vagus nerve or affection of the sinus node. We have often observed this phenomenon during heart surgeries.

In the electrocardiogram in Figs. 23a and 25a, there is noted a periodic deviation of the sinus and nodal P waves during a number of cycles; various transitional shapes are detected among these shapes. These transitions sometimes occur parallel to the respiratory phases. In the case of a

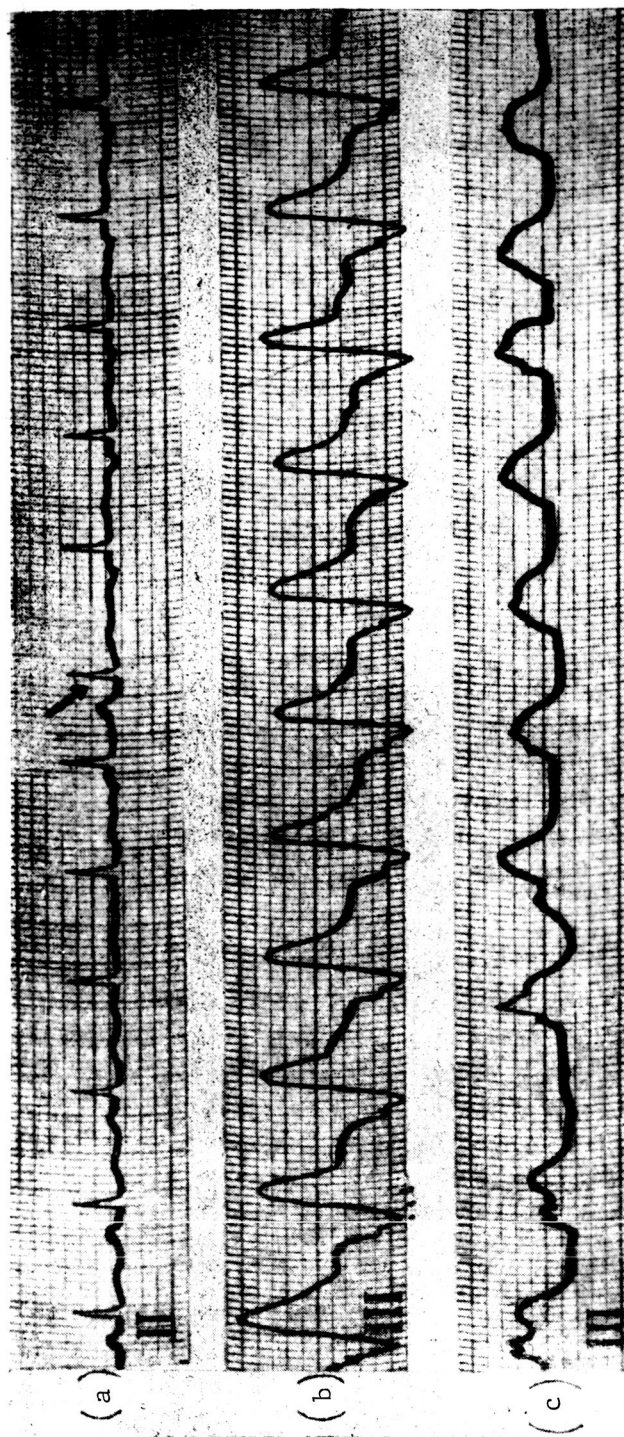


Fig. 25: Impairment of the automatic process. (a) Electrocardiogram of a patient, with the following diagnosis: mitral defect with prevalence of stenosis. Migration of the cardiac pacemaker resulting in an extrasystole from the right auricle (indicated by the arrow), and the beginning of an upper nodal rhythm. (b) Electrocardiogram of a patient, B. O., 31 years old, with the following diagnosis: mitral defect with prevalence of stenosis. The intraventricular rhythm is of the ventricular tachycardiac type (during the time of the mitral commissurotomy), (c) Electrocardiogram of the same patient. Individual ventricular contractions of the monophasic-curve type after heart massage; lastly the stopped heart of the patient.

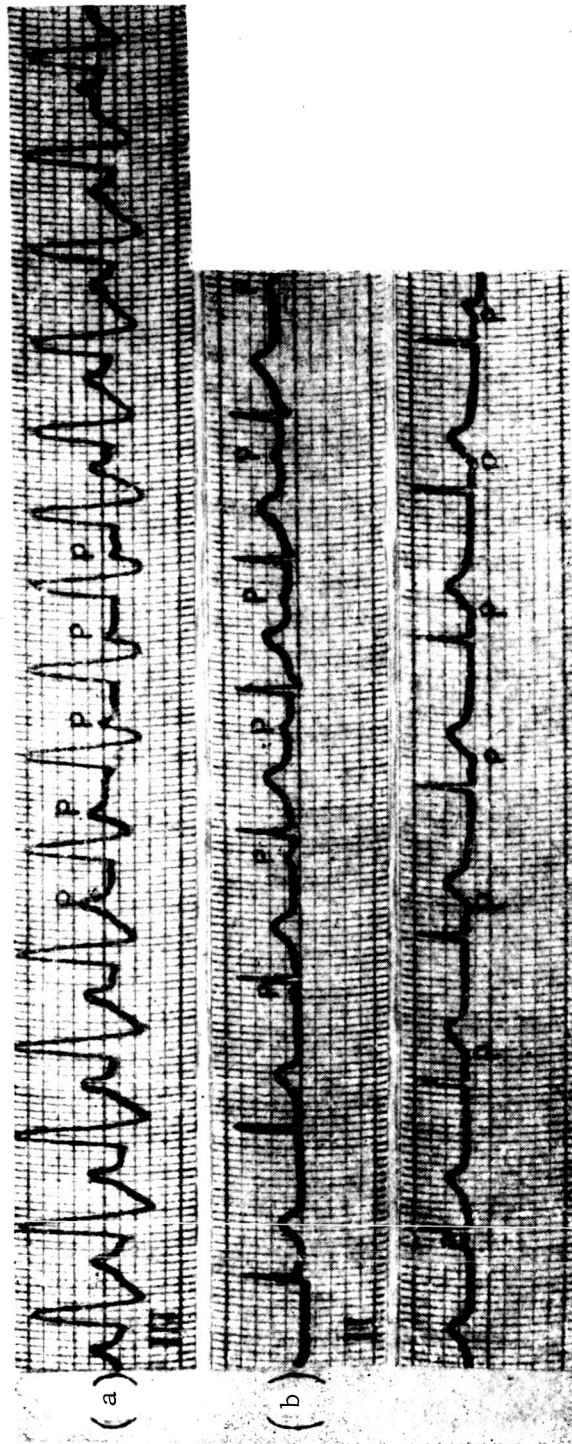


Fig. 26: Migration of the pacemaker of cardiac rhythm. (a) Electrocardiogram of a patient, B. O., 31 years old, with the following diagnosis: mitral defect with prevalence of stenosis. The P-Q segment now contracts and expands in accordance with the change in the shape of the P wave. (b) Electrocardiogram of the patient, T. M., 9 years old; the study was made for the purpose of obtaining a more precise diagnosis. On the given fragment, the rhythm emerges from the middle part and then enters into the sinus node. The pacemaker of the rhythm then migrates once more into the atrioventricular node down to its lower part and, accordingly, the shape, location of the P wave, and length of the P-Q segment change. (The bottom diagram is the direct prolongation of the diagram above).

patient suffering from scleroderma we observed a migration of the pacemaker with a simultaneous change in the rhythm of the cardiac contractions. The rhythm was relatively reduced during the period of sinus formation. During the period of nodal formation its frequency was increased. Chevalier and Fisch (1960) described an interesting phenomenon during which a migratory pacemaker acts in the atrioventricular node and a "dual" nodal tachycardia is observed.

Still other types of migrations of the pacemaker of cardiac rhythm are encountered. In the usual migration from the sinus to the atrioventricular node the P-Q segment gradually contracts and on the reverse pathway it gradually expands (the length of the segment does not change during the migration of the pacemaker within the sinus node). We observed a case where during the mitral commissurotomy the P-Q segment in the course of a number of cycles expanded at one time and contracted at another time (Fig. 26a). It is interesting to note that in the case of this patient there later was observed an upper nodal rhythm changing into a ventricular rhythm with subsequent cardiac arrest. The gradual contraction of the P-Q segment during the course of a number of subsequent cycles down to the point of its disappearance was observed in dogs during experimental reproduction of a complete atrioventricular block. In one of the observations we made, the migration of the pacemaker took place from the sinus to the lower part of the atrioventricular node (Fig. 26b). We assume that in all of these cases there is also a kind of disruption of the atrioventricular conductance as a result of which the P-Q segment periodically contracts or expands.

c. Autonomous auricular rhythm. In this case two centers of excitation operate simultaneously: a sinus center (the pacemaker center) and the auricular center. On the electrocardiogram, together with the usual auricular complexes which are followed by the ventricular complexes there are observed auricular waves of another configuration with less frequent rhythm and without the subsequent ventricular complexes. One case which we studied is of interest: after each impulse from the auricular center the pacemaker of the cardiac rhythm migrated at once to the lower part of the atrioventricular node and then, during the course of a few complexes, gradually increased in the sinus node (Fig. 26a). In the case of this patient a slowing down of intra-auricular conductance took place.

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V. Ye. Nezlin and S. Ye. Karpay (1959) associated the autonomous auricular rhythm to the intra-auricular block. A new autonomous center arises in the determined blocked zone of the auricle.

d. Intraventricular rhythm. The rhythm of the heart comes from one of the automatic centers of third degree, from the common limb of the bundle of His, or from one of its stems, and sometimes from all of these centers (F. Ye. Ostapyuk, 1958). This rhythm is encountered rather infrequently, especially in those forms in which the auricle is excited by the ventricular impulse. More often, a short-lived appearance of this rhythm is observed.

On the electrocardiogram there is detected an expression of bradycardia

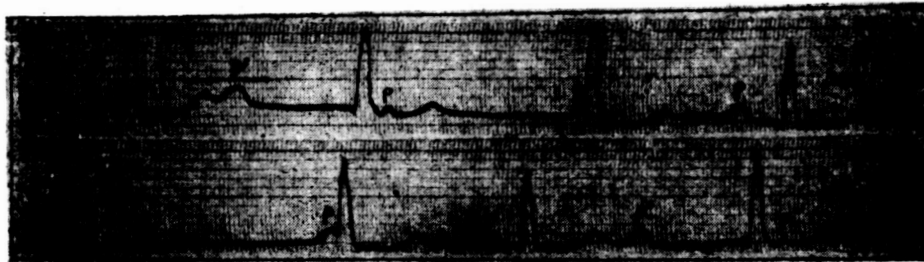


Fig. 26a: Impairment of the automatic process. Electrocardiogram of a patient, B. K., 14 years old, with the following diagnosis: tetralogy of Fallot, autonomous auricular rhythm, P wave from the auricular center (explanation in the text). The lower diagram is the direct prolongation of the diagram above.

and ventricular complexes with the characteristic pattern of partial dependence upon the ventricle from which the rhythm comes (see page 59). Retrograde P waves may or may not appear, since they are superimposed on the ventricular complexes. In heart operations, we sometimes observed a ventricular rhythm with great frequency which can be included in the group of ventricular tachycardias (Fig. 25b). In serious cases, less frequent ventricular fluctuations were observed preceding cardiac arrest or arising after the heart had been massaged (Fig. 25c).

3. Disturbances in Conductivity

/100

The various impairments in the functions of conduction (or block) can be partial when the passage of the impulse is slowed down in a given part of the conductor system, and total when there occurs a complete cessation of the passage of the impulse through this section. Blocks are observed in an increased tone of the vagus nerve, in the activity of various toxins and medications (in particular in the case of digitalis intoxication), and in such diseases as rheumatism, cardiosclerosis, and syphilis. Still other congenital forms of complete block have been described, for instance a substantial block of the left limb of the bundle of His as occurs with a congenital insufficiency in blood supply to the diaphragm (Deforest, 1956). A complete atrioventricular block has been described in the case of a healthy aviator (Mathewson and Harvie, 1957).

There are the following types of blocks:

A. Sinoauricular block. In this case an alternate impulse in the sinus node either is not formed, or it does not pass into the auricle, resulting in a periodic omission of one of the cardiac contractions. A block of this type can only be partial since a total sinoauricular block is fatal if some other center does not take over the automatic function.

In the electrocardiogram in Fig. 27a, direct phenomena for this type of

block are not observed since the excitation process in the sinus node and its passage from the sinus to the auricles do not appear on the electrocardiogram even though Van der Kooi et al. (1956) were able to record the local excitation in the sinus (and atrioventricular) nodes during experiments on dogs. This block can be recognized by the appearance of a long pause which is equal to two cycles of the heart or which is somewhat less than this interval because the impulse following the block passes to the auricle relatively more rapidly. The sinoauricular block may take place in a regular sequence: 1:1, 3:1, 5:1 and so forth, or else without any sequence. In the case of block 1:1 it is necessary to differentiate it from the usual sinus bradycardia. If we have a 1:1 sinoauricular block, by administering atropine or applying some physical strain, the number of heart contractions initially doubles and then some increase in the frequency of the rhythm is observed. In the case of a typical sinus bradycardia these tests only result in a gradual increase in the frequency of cardiac activity.

A peculiar aspect of this block is that of sinus arrest whereby the sinus node ceases to function, and the ventricles are excited by the impulse from the automatic centers of the second or third degree. Myers (1956) considers this phenomenon to be the manifestation of a syndrome of the carotid /101 reflex and Katz and Pick (1956) associate this with the effect of great doses of quinidine. Still another phenomenon is encountered--auricular standstill--in which case the electrical activity of the auricle ceases, and recorded on the electrocardiogram are ventricular complexes having a normal sequence, but without the preceding P wave. In our observations during surgery for mitral or aortal stenosis we came upon such cases, but we experienced difficulty in differentiating them from the usual nodal rhythm.

B. Intra-auricular block. This type of block is partial and is associated with the morphological changes or the hypertrophic processes in the auricular musculature, for instance in the case of mitral stenosis or cardio-sclerosis. On the electrocardiogram augmented P waves are observed with various changes in their shapes (Fig. 40).

C. Atrioventricular block. These are found in two different types: complete block and incomplete block.

a. Incomplete atrioventricular block. A number of types have been observed. The first type of incomplete atrioventricular block is encountered rather frequently and is characterized by a slowing down of the atrioventricular conductance, but each sinus impulse does reach the ventricles. In the electrocardiogram in Fig. 27b an increase in the P-Q segment is noted. In pronounced cases this increase may extend up to 0.30 - 0.40 sec. In the case of a rather sharp increase in the P-Q segment the P wave flows into the T wave of the preceding complex and definite difficulties in differential diagnosis arise.

The second type of incomplete atrioventricular block is characterized by the appearance of Wenckebach-Samoylov periods. In this case the passage of each of the successful impulses through the atrioventricular node gradually

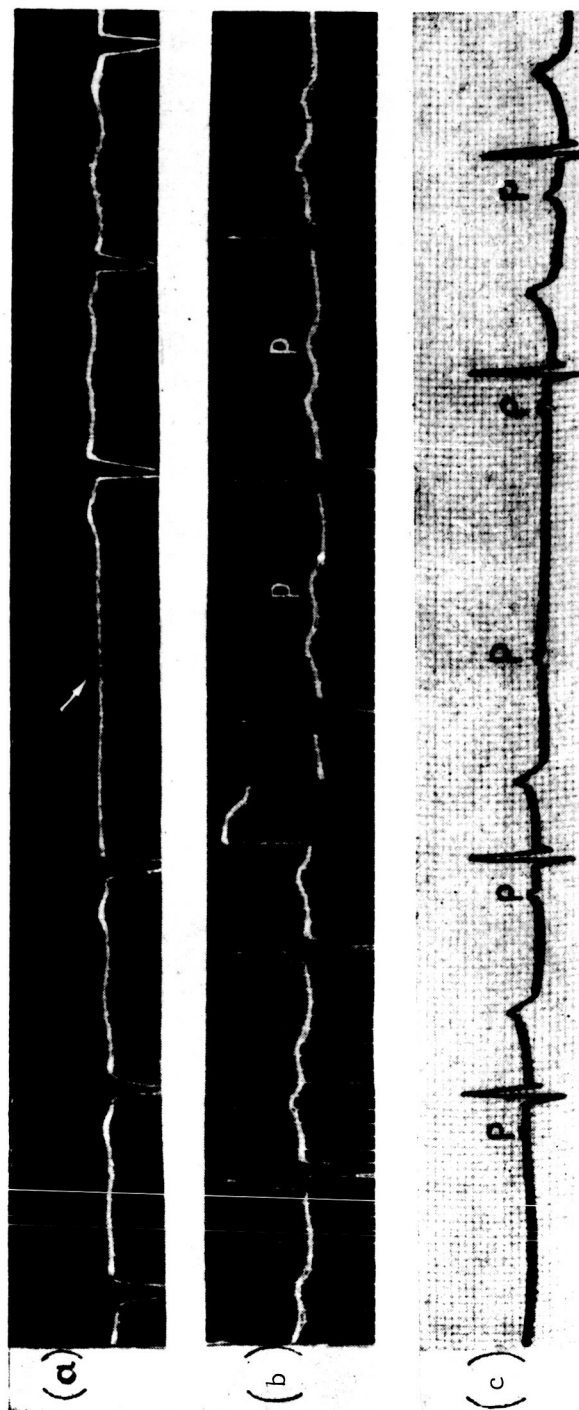


Fig. 27: Impairment of the conduction. (a) Electrocardiogram of a patient, A. B., 69 years old, with the following diagnosis: atherosclerotic cardiosclerosis. Sinoauricular block. The arrow indicates the omission of a complete cardiac complex. (b) Electrocardiogram of a patient, S. K., 53 years old, with the following diagnosis: atherosclerotic cardiosclerosis. Incomplete atrioventricular block, of the first type, segment P-Q = 0.29 sec. (c) Electrocardiogram of a dog. Incomplete atrioventricular block with a Wenckebach-Samoylov period. In the case of the dog, at one time the second type was observed and at another time the third type of incomplete atrioventricular block was observed. The time marks in illustrations (a) and (b) are 0.05 sec.

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slows down and when the deterioration of the conductance reaches a certain degree, the next impulse cannot pass through the atrioventricular node, the ventricles are not excited and their contraction ceases. A pause is created during which the atrioventricular conductance is reestablished and the recorded regularity begins once again.

On the electrocardiogram in Fig. 27c, there is observed a gradual increase in the P-Q segment during the course of a few cardiac cycles and then the Wenckebach-Samoylov period sets in and a P wave is obtained without the next ventricular complex. These cycles repeat themselves regularly and thus, depending upon the functional condition of the atrioventricular node, each fourth impulse, fifth impulse, and so on, may disappear.

The third type of incomplete atrioventricular block is characterized only by a periodic disappearance of the ventricular contractions. On the electrocardiogram in Fig. 27c, the P-Q segment does not change (or is somewhat larger), but, with the definite regularity, the P wave is recorded without the subsequent ventricular complex. We observed a rare case: disappearance of the /103 ventricular contraction took place with a 1:1 frequency (Fig. 28a).

There is no uniform opinion among authors concerning the development of these types of incomplete atrioventricular blocks since the very mechanism of atrioventricular conductance is not definitely clear. In particular, there are many different aspects for explaining the appearance of the Wenckebach-Samoylov period. It is interesting to note that Copeland and Stern (1958) gave particular significance to humoral factors in the genesis of this phenomenon.

Obviously, there are still other types of incomplete atrioventricular blocks. We propose that individual types of cardiac pacemaker migrations (see page 96 of the original text) can be treated as being original types of incomplete atrioventricular blocks.

b. Complete atrioventricular block. The sinus impulses arise regularly, but, as a result of some obstacle occurring at the level of the node or a common limb of the bundle of His, they do not enter the atrioventricular system. Thus, the auricles contract with the sinus rhythm and the ventricles receive impulses from the node or even from the lower parts of the atrioventricular system.

On the electrocardiogram in Fig. 28b, c, alternating complexes appear regularly one after the other with the usual sinus rhythm. Also, ventricular complexes appear regularly following one after the other with a slow nodal rhythm, or even a less frequent rhythm, if the impulses arise from the lower parts of the atrioventricular system. There is no regularity in the appearance of the auricular and ventricular complexes, and they may even occasionally disappear. If the ventricular impulse arises in one of the limbs of the bundle of His, then aberrant complexes appear (Fig. 28b). In the remaining cases the ventricular complexes have the usual configuration.

In the case of a strongly advancing atrioventricular block or in the

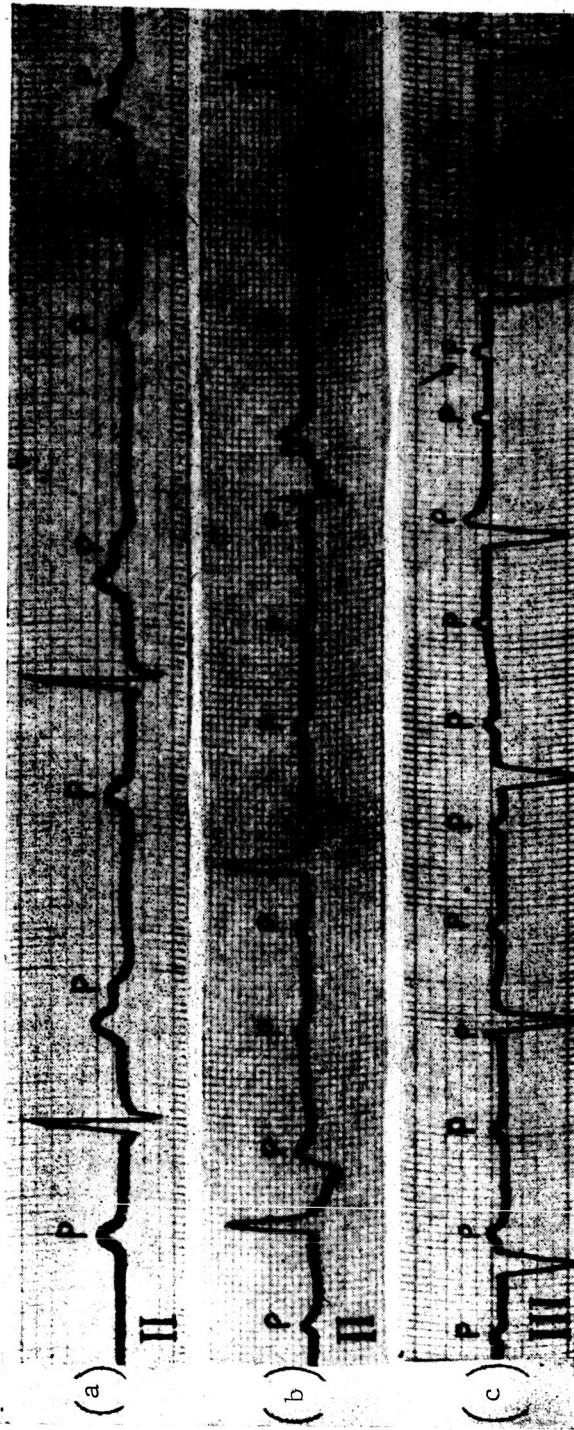


Fig. 28: Impairment of the conduction. (a) Electrocardiogram of a child patient M. G., 10 years old, with the following diagnosis: congenital defect of the heart (indistinct type). Incomplete atrioventricular block, of type III, with disappearance of the ventricular complex of each second contraction; the P wave overlaps onto the T wave of the preceding complex resulting in the presence of sinus tachycardia and significant slowing down of the atrioventricular conductance, the P-Q segment equals 0.20 sec. (b) Electrocardiogram of a dog during an experiment with artificial reproduction of the block. Complete atrioventricular block after inserting a suture (commissure) onto the area of the node. The ventricular impulses develop from various points of the atrioventricular system. (c) Electrocardiogram of a patient, M. K., 58 years old, with the following diagnosis: atherosclerotic cardiosclerosis, complete atrioventricular block. The auricular wave, indicated by an arrow, apparently corresponds to the sinus extrasystole.

case of a sharply defined bradycardia, the Morgagni-Adams-Stokes syndrome appears as the result of cerebral hypoxemia. I. M. Rezvin (1958) detected 14 cases of this syndrome in patients with complete atrioventricular block. Palmer (1956) described an interesting case when these attacks, during complete atrioventricular block, emerged as a result of the periodic retardation and cessation of the rhythm of impulses from the ventricular center.

D. His bundle branch block. In this form of block the passage of the impulse is blocked in one of the branches of the bundle of His which results in a given ventricle below the block receiving excitation from the adjacent ventricle through the entire thickness of the intraventricular diaphragms. A high degree of clarity was attained concerning the problem of the morphology of the ventricular complexes in the case of this block by means of experimental observations which showed that by cross-cutting the right branch of the bundle of His we obtain a so-called partial pattern of the left ventricle, and by cross-cutting the left branch of the bundle of His we obtain a partial pattern of the right ventricle (see page 59 of the original text). The first type of curve is observed during a block of the right branch of the bundle of His, the second type of curve is observed during a block of the left branch of the bundle of His. It should be kept in mind that in the case of the blocks the pattern of the electrocardiogram also depends upon such factors as the position of the heart in the chest, the accompanying disease of the myocardium or hypertrophy of the ventricles. /105

In the case of a block in the left branch of the bundle of His (Fig. 29) there is observed in lead I a wide, high and split R which is displaced below the RS-T segment and a wide negative T wave. A reverse pattern is recorded in lead III. In the right precordial leads there are obtained ventricular complexes of the QS type, or, less frequently, of the rS type, the RS-T segment has an upwards deviation and the T wave becomes positive. In the left precordial leads there is observed a high, wide and split R wave, the Q wave is absent, the RS-T segment has an upwards deviation and the T wave is negative. In these positions the time of formation of the internal deviation fluctuates within the range of from 0.07 to 0.09 sec. During a vertical position of the heart there is obtained in lead aVR a ventricular complex of the QS type and T is positive. Approximately the same pattern is observed in lead aVL and in lead aVF a high and wide R wave develops with a negative T wave. During horizontal position of the heart, there appears in lead aVR a ventricular complex of the QS type, T is positive. In lead aVL there is observed a high and wide R wave, T is negative, and in lead aVF there is recorded a complex of the rS type and the T wave is positive.

During a block of the right branch of the bundle of His (Fig. 30) there is observed in lead I a complex of the rS type with a wide and notchy S wave, the T wave is positive and in lead III the complexes present a reverse pattern. In the right precordial leads a complex of the rSr' or rSR' type is recorded, the time of formation of the internal deviation is prolonged up to 0.07 sec., and the T wave is negative. In the left precordial leads the ventricular complexes have a qRS shape with a wide and notchy S wave. The T wave is positive. /106

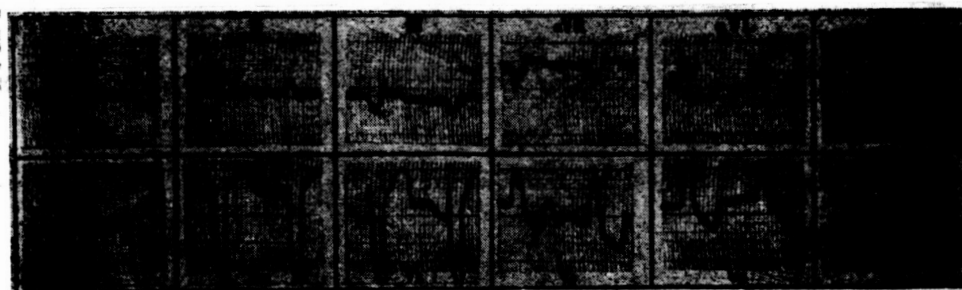


Fig. 29: Impairment of the conduction. (a) Electrocardiogram of the patient, T. V., 64 years old, with the following diagnosis: hypertension, stage IIb; left bundle branch block.

We have cited the classical forms of blocks in the branches of the bundle of His. In practical work many variants are encountered, among which the most frequent are the incomplete forms of blocks. Characteristic for an incomplete block of the left branch of the bundle of His is the widening of the QRS complexes to no greater a period than 0.12 - 0.13 sec., and in standard leads the presence of the R wave without the q wave in the left chest positions with a formation time of the internal deviation of from 0.05 to 0.08 sec. In the case of an incomplete block of the right branch of the bundle of His (Fig. 43) the QRS complex in the standard leads has a width which is no greater than 0.12 - 0.13 sec. and in the right precordial leads, complexes of the rR', rsR' and rSR' types are encountered with a formation time of internal deviation of from 0.04 to 0.07 sec.

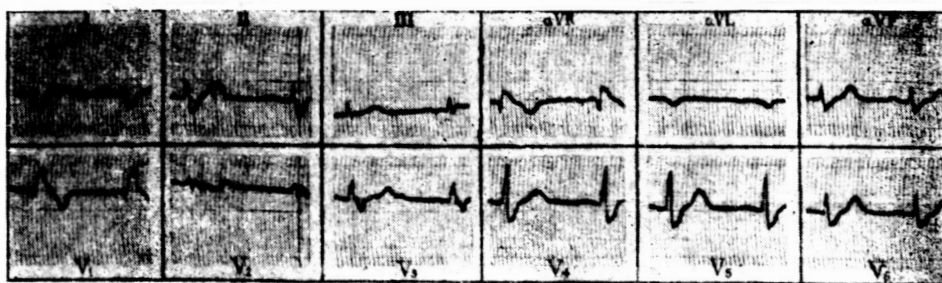


Fig. 30: Impairment of the conduction. Electrocardiogram of the patient A. S., 36 years old, with the diagnosis: Myocardial cardiosclerosis (?); right bundle branch block.

E. Bundle branch block and terminal branch blocks. This type of block is observed during significant diffuse changes in the myocardium of the ventricles when the entire ventricular conductor system including the Purkinje network is impaired. On the electrocardiogram, significant changes are observed in the QRS complexes, for example, they are sharply decreased, deformed, widened, and the T waves are sharply decreased or they are negative.

F. Syndrome of premature excitation of the ventricles (WPW syndrome).

This syndrome, which is also designated as an aberrant atrioventricular conductance, abnormal atrioventricular conductance, and so forth, were described in 1930 by Wolff, Parkinson and White and in their honor they are still called the WPW phenomenon (the first letters of the last names of these authors). It is found in all ages and is detected in 1.5 out of a thousand patients /107 (Hejtmancik and Hermann, 1957). The essential feature of the phenomenon lies in the fact that the P-Q segment is contracted (according to Katz and Pick (1956), it is less than 0.12 sec.), and there is an enlarged and deformed QRS complex, found particularly in its initial section where there is observed the so-called delta wave (a small and notched fluctuation). Sometimes, the RS-T segment and the T wave appear discordant, depending upon the changes in the QRS complexes. In this syndrome, there often appear attacks of supraventricular tachycardia and during the time of paroxysm the phenomenon disappears. It also disappears under the effect of such pharmacological agents as atropine and amylnitrite.

Various opinions are brought forth in the literature concerning the genesis of the WPW phenomenon. A number of authors propose that the sinus impulse does not reach the ventricles along the usual pathways but along the Kent bundle, or along other fibers. Because of this, the time of the atrioventricular conductance also decreases significantly. According to this theory, the widening of the QRS complex is explained by the fact that the impulse reaches the right ventricle earlier, and from there it spreads into the left ventricle. A case which we observed supports this theory. During a mitral commissurotomy (with trans-ventricular access), the pacemaker of the WPW syndromes disappeared as soon as the instrument was taken away from the cavity of the left ventricle (Fig. 31a). We assume that there apparently took place a temporary compression, by means of the atrioventricular node or the common branch of the bundle of His. Fox (1957) discussed the presence of an additional conductor pathway between the auricle and the ventricle, but he also lends importance to vagotonia. L. I. Vogelsson (1957) found that during the WPW phenomenon the automatic process of the sinus node is suppressed, and the rhythm arises from any point in the vicinity of the atrioventricular node. The author relates the widening of the QRS complex to the unstable disruption of the intraventricular conduction. V. Ye. Nezlin and S. Ye. Karpay (1959) proposed that the essence of this entire problem is the change in the functional condition of the atrioventricular node. Supporting the opinion of V. Ye. Nezlin is another case which we studied in which, during the time of surgery for mitral stenosis, there was first observed a WPW syndrome and then an upper or medium atrioventricular rhythm (Fig. 31b). R. P. Stambolitsyan (1957) found that this syndrome is encountered during functional disruptions of the central nervous system, and the extracardial nerves play a significant role in its genesis. The observation made by Lamb in 1958 also discussed this. In this experiment the syndrome disappeared under the effect of each inspiration and reappeared during the expiration. We believe that in the genesis of the WPW syndrome a role is played by all of the mechanisms indicated but that in each individual case one of these mechanisms is the prin-

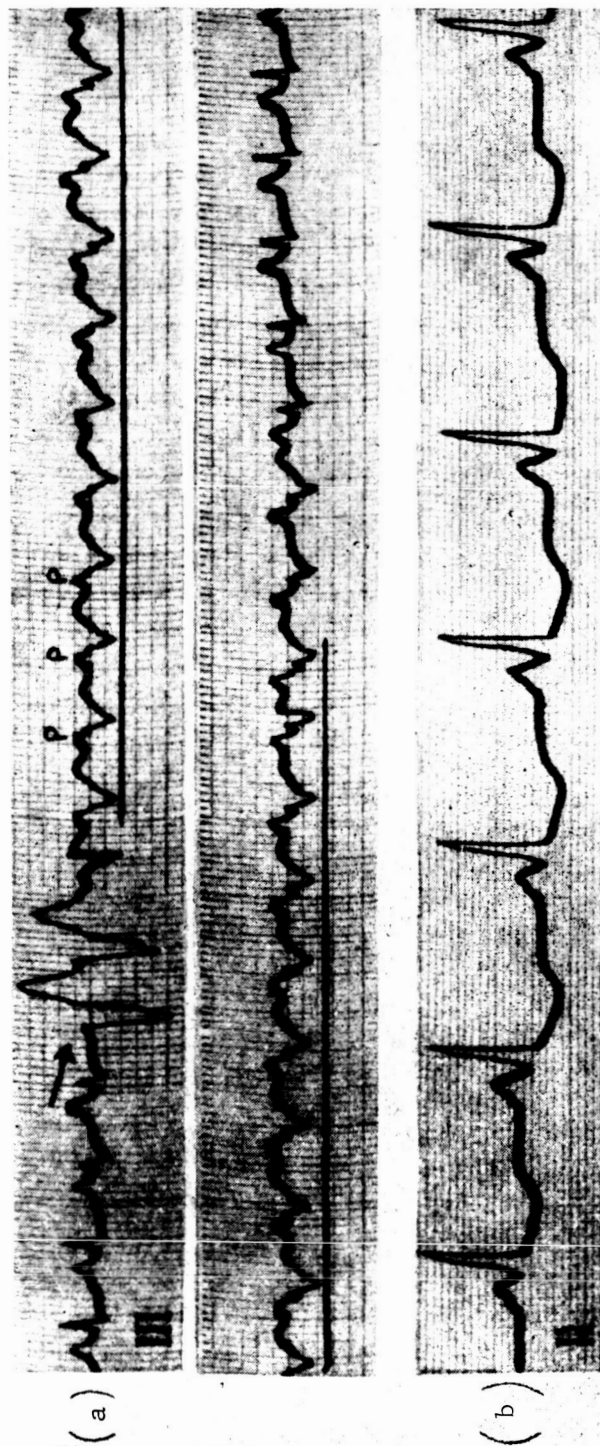


Fig. 31. Premature excitation of the ventricles. a) Electrocardiogram of patient B.L., age 22, with a diagnosis of mitral stenosis. During the interventricular commissurotomy grouped ventricular extrasystoles appear (indicated by arrows), followed by the WPW syndrome (underlined), which disappears upon removal of the instrument from the left ventricular cavity (2nd picture is a continuation of the first). b) Electrocardiogram of patient G. A., age 25, with a diagnosis of mitral stenosis. An alteration of the atrioventricular rhythm, the sinus rhythm and the WPW syndrome were observed. The above fragment shows the WPW syndrome.

cipal actor. It is interesting to note that Averill (1956) attaches some importance to the hereditary factor, too.

4. Disturbance in Excitability

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A. Extrasystolic arrhythmia. The term extrasystole implies a premature beat of the whole heart or its individual sections. The other terms suggested for that phenomenon--premature beat, ectopic beat, and premature systole--are possibly more descriptive. In this study, however, we will use the word extrasystole which is the commonly accepted term in medical literature.

An extrasystolic impulse may originate from any section of the heart. The diastole following such a beat usually lasts longer than the diastolic pause between two normal beats. This is referred to as a full compensatory pause if the total duration of the interval between the two normal contractions surrounding the extrasystolic complex equals the total duration of two normal cardiac cycles. A gull compensatory pause occurs when the extrasystolic impulse fails to discharge the current sinus impulse which comes into being but is not seen, or rather not detected, on the electrocardiogram. If the extrasystolic impulse discharges the sinus node, the result is a shortened compensatory pause, and the closer the focus of extrasystolic irritation to the sinus node, the shorter the pause.

Extrasystolic arrhythmia is observable in a large variety of physiological and pathological states as well as in various cardiac operations. There is no unanimity of opinion on its genesis. Some authors assume the occurrence of a reentry phenomenon when the impulse is blocked in some section of the myocardium because of the local extension of the refractory period, and after a certain delay it passes in a different direction and, finding the heart in a nonrefractory phase, excites it prematurely. Other authors believe that the extrasystole is accompanied by the existence of a latent focus whose impulses stimulate the heart whenever there are favorable conditions that tend to intensify cardiac excitability. Related to this is the parasystole theory according to which a permanent active focus functions in some small section of the heart; that focus cannot be detected, as the particular section is very small, and its potential very insignificant. Under favorable conditions one of the impulses of this focus extends to the entire heart and produces a premature contraction. The genesis of an extrasystole can be successfully explained also on the basis of the N. Ye. Vvedensky theory of the electrical properties of a parabolic focus. We believe that the mechanism governing the formation of an extrasystolic impulse cannot be uniform, and that different mechanisms are operative under different conditions. It should/110 also be emphasized that the dynamic state of the extracardial nerves plays an important part in all these mechanisms.

The extrasystoles are divided into sinus, precordial, atrioventricular and ventricular, according to the localization of the premature impulse focus.

A sinus extrasystole is indicated on an electrocardiogram by a premature cardiac complex with the usual configuration. The P-Q segment of the

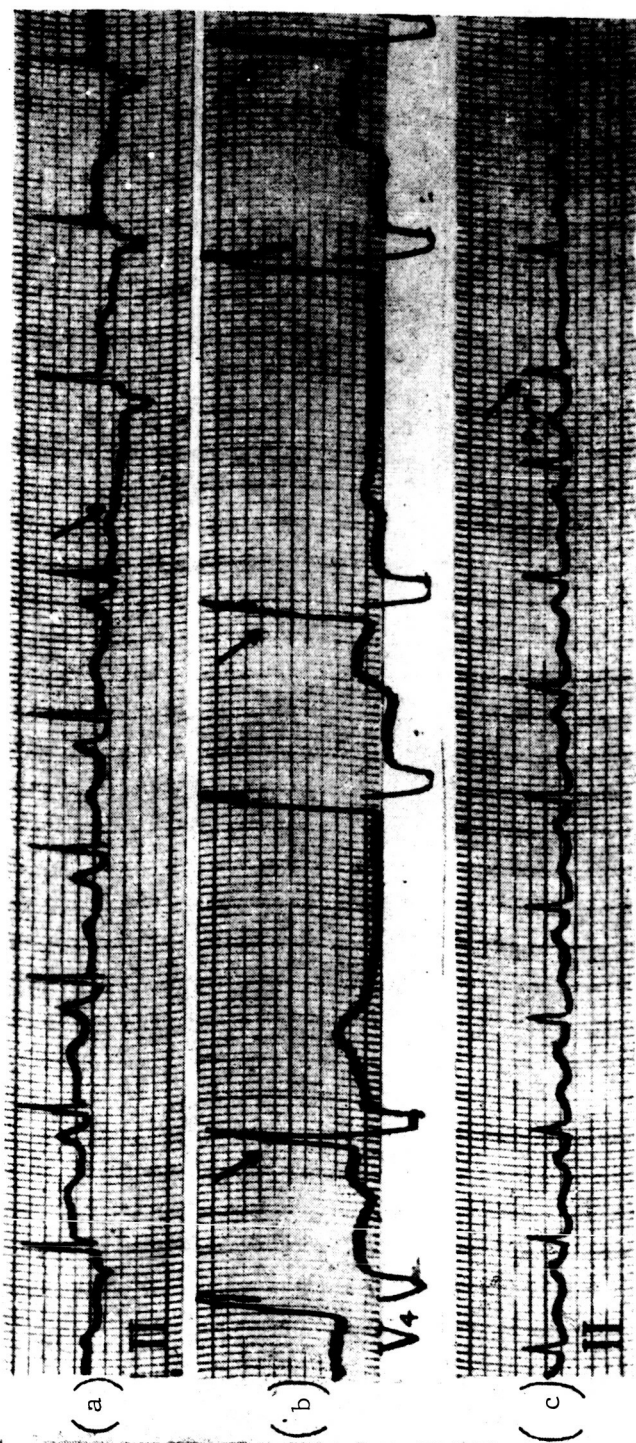


Fig. 32. Extrasystolic arrhythmia (the extrasystoles are indicated by arrows). (a) Electrocardiogram of patient P. E., aged 21, with the following diagnosis: mitral defect with prevalent stenosis. A blocked sinus beat (?) followed by the development of an upper nodal rhythm. (b) An electrocardiogram of patient Sh. F., aged 68, with the following diagnosis: hypertension, stage IIb. Extrasystoles from atrium dextrum of the bigeminy type. (c) Electrocardiogram of patient K. A., aged 27, with diagnosis: mitral defect with prevalent stenosis. Extrasystole from atrium dextrum and subsequent systole emerging from the central part of the atrioventricular node.

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extrasystolic complex may occasionally be somewhat shortened. The pause following the extrasystolic complex is of the usual duration so that there is no compensatory pause.

A premature beat with the usual ventricular complex but a changed P wave is observable in the case of a precardiac extrasystole (Fig. 32c). If the impulse originates from the upper sections of the atrium dextrum, the P wave resembles a somewhat altered sinus wave; if the impulse comes from its lower sections, it extends retrogressively to the atrium cordis, and the result is a P wave with a reverse polarity. The P-Q segment may fluctuate within the norm or appear somewhat increased, especially in case of a highly premature impulse. An impulse originating from the atrium sinistrum is indicated by the appearance of negative P waves. In a precordial extrasystole the compensatory pause is relatively longer but not as long as a full pause.

A premature beat with a usual ventricular complex is observable in the case of a nodal extrasystole (Fig. 33 a & b). An impulse originating from the upper section of the atrioventricular node is indicated by a nodal P wave with a shortened P-Q segment. If the impulse originates from its central section, the P wave is missing, and if it comes from the lower section, the nodal wave is recorded between the QRS complex and the T wave. The compensatory pause is then close to a full pause, and it may occasionally be equal to a full pause when the extrasystole originates from the lower section of the node or the general stem of the bundle of His.

In the case of the above-described supraventricular forms of extrasystole, the ventricular complex of the extrasystolic beat frequently reveals certain changes in the form of a downward shift of the RS-T segment, and a reduction of the T wave amplitude in relation to the other complexes that are usual to this lead. These changes are caused by the inadequate nutrition of the myocardium as a result of the reduced pulsating volume of the heart by the shortened period of hyperemia. The complex following the extrasystole, on the other hand, acquires a more normal or "healthy" appearance as the cardiac beat occurs in conditions of good hyperemia because of the lengthy compensatory pause.

In the case of a ventricular extrasystole, the impulse originates from /112 the branches of the bundle of His or some section of one ventricle and, exciting that ventricle, extends to the neighboring ventricle through the muscular layer of the ventricle, particularly through the interventricular septum. This case is characterized by the appearance of ventricular complexes with a morphology similar to that of the complexes produced by a blockade of the branches of the bundle of His: high, wide, split and discordant. The P-wave is missing, having fused with the QRS complex; the extrasystolic impulse from the ventricle usually retrogresses to the atrium cordis but reaches it only when the sinus node has already released its next impulse, and the result is an interference between the ectopic and sinus impulses. Such a picture leads to a full compensatory pause of the ventricular extrasystole.

In the case of a dextroventricular extrasystole (Fig. 33c), in lead I the characteristic ventricular complexes reveal a maximum positive fluctuation, and T is negative; in lead III a maximum negative fluctuation in QRS complex and a positive T are observable. In the right precordial lead, the

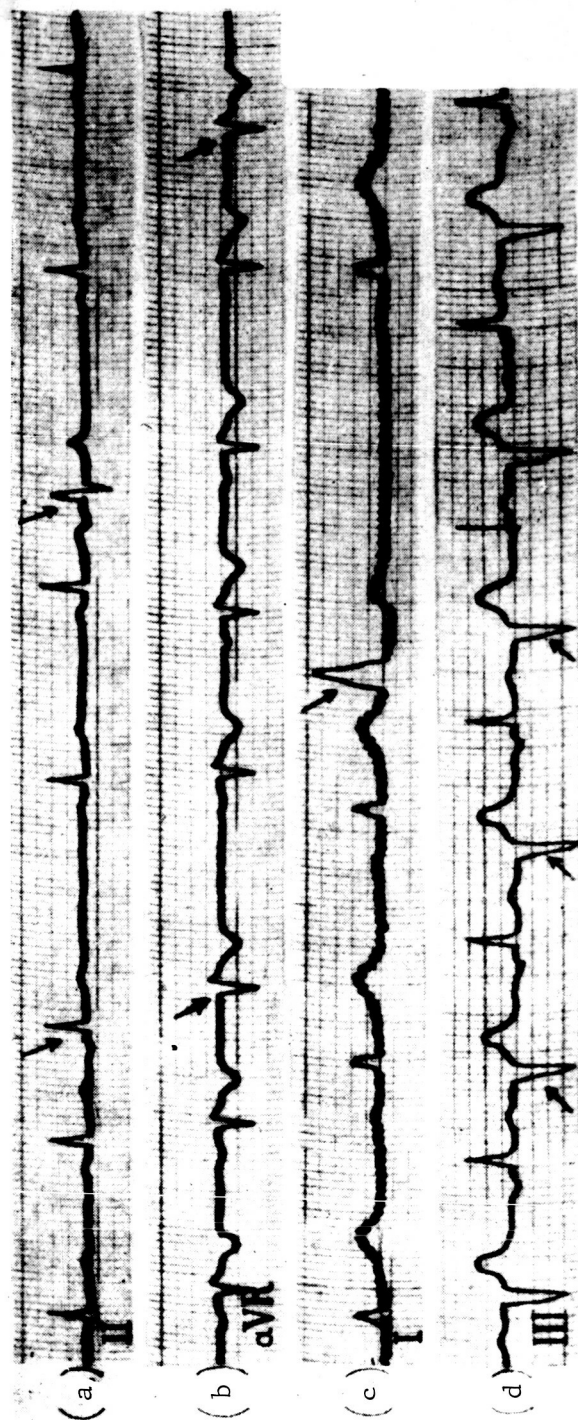


Fig. 33. Extrasystolic arrhythmia. (a) Electrocardiogram of patient G. S., age 22, with following diagnosis: struma II without thyrotoxicosis. Two extrasystoles from the upper part of the atrioventricular node: ventricular complex of the second premature contraction of aberrant organs. (b) Electrocardiogram of patient S. G., aged 37, with following diagnosis: mitral defect with prevalent stenosis. Two extrasystoles from central part of atrioventricular node. (c) Electrocardiogram of patient G. F., aged 23, with following diagnosis: active rheumatism and mitral defect. Extrasystole from right ventricle. (d) Electrocardiogram of patient A. P., aged 52, with following diagnosis: atherosclerotic cardiosclerosis. Ventricular extrasystoles of the bigeminy type and upper nodal rhythm.

ventricular complexes are of the rS or QS type, and a high R wave with a considerably longer time of internal deviation is noted in the left lead.

In the case of a left ventricular extrasystole (Fig. 34), in lead I the typical ventricular complexes are characterized by a maximum negative fluctuation, and T is positive; in lead III a maximum positive fluctuation of the ventricular complexes is observable and T is negative. Noted in the right precordial leads are complexes with a maximum fluctuation, and a considerable increase in the time of the origin of internal deviation, while Rs-shaped complexes are found in the left precordial positions.

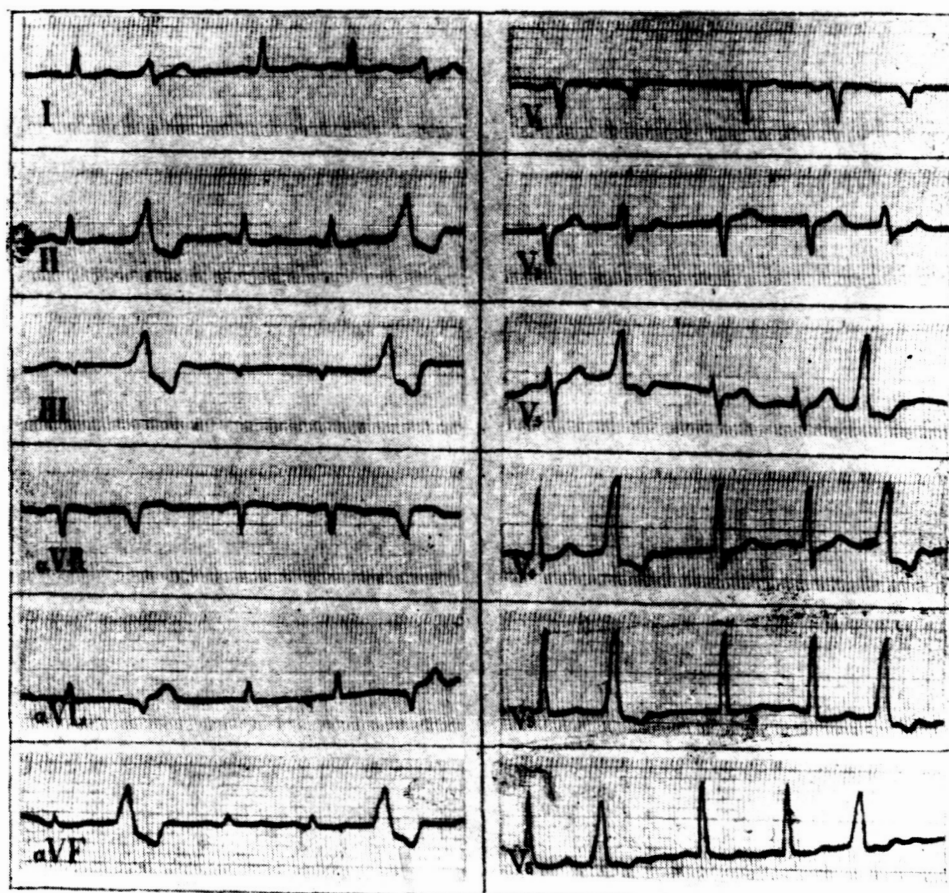


Fig. 34. Extrasystolic arrhythmia. Electrocardiogram of patient S.B., age 49, with following diagnosis: hypertension, Stage IIb; trigeminal type extrasystole from left ventricle.

Ventricular extrasystoles originate from the basis cordis and from the apex of the heart. In the first case, a positive fluctuation in the QRS complex is predominant in all the standard leads, and the T waves are more often negative. Ventricular complexes with a maximum positive fluctuation are also recorded in the precordial leads. If the extrasystoles stem from the apex of the heart, the ventricular complexes in all the three standard leads are characterized by a maximum negative fluctuation, and the T waves

are frequently positive. A similar picture is observable also in the precordial leads from V_2 or from V_3 to V_6 .

All the above-described extrasystoles may be divided into individual, frequent and group-type (Fig. 35 a & c). They may originate without any definite regularity or with a definite rhythm: after every other normal contraction (bigeminy) (Figs. 32b, 33d and 35b), after every two normal contractions (trigeminy) (Fig. 34), etc. The impulses may originate from one or several different leads (polytopic extrasystoles (Fig. 35a)). The precordial extrasystolic impulse is occasionally blocked in the atrioventricular node, the ventricles are not excited, the premature contraction is shown on the electrocardiogram as having only a P wave, and the ventricular complex is missing. A lengthy pause may lead to the formation of an escaping systole from the atrioventricular node or, less often, from some other section of the ventricles (Figs. 32c and 35c). /114

There is also a special type of extrasystole which is much better described by the term extrasystole. It is called an interpolative extrasystole, and is characterized by a premature contraction (primarily ventricular) in the normal interval between two usual contractions (Fig. 36a). Interpolative extrasystoles are observable whenever the diastole is fairly lengthy and the premature impulse occurs very early. It may be pointed out that the P-Q segment of the post-extrasystolic contraction is usually increased. /116

One of the variations of the extrasystole is the parasystole. In this case there are two simultaneously functioning foci: one is a normal sinus and the other an ectopic focus which is frequently precordial and less often ventricular. The sinus impulse cannot penetrate into the parasystolic focus because of the blocked entrance, and this results in the periodic formation of extrasystoles. These are regularly reflected on the electrocardiogram at definite time intervals. The intervals between two neighboring extrasystoles should be the same, although if they differ, they still have a common divider. There is no correlation here between the extrasystolic complex and the individual components of a normal contraction. This phenomenon has no particular pathological significance.

Another variation of the extrasystole, which is relatively rare and little different from the usual extrasystole, is the fusion beat. In this phenomenon the sinus impulse excites the atrium cordis, and as the impulse enters the system of the bundle of His, the ventricles are excited by an ectopic ventricular impulse. A cardiac complex with the usual sinus P-wave and a changed QRS ectopic complex thus appear on the electrocardiogram (Fig. 36 b & c); the P-Q segment is shortened.

There is still another variation of an extrasystole which is also rare and has no independent pathological significance. The point under consideration is reciprocal beats, that is, when the nodal or, less often, ventricular premature impulse extends retrogressively to the atrium cordis and then returns to the ventricles stimulating them a second time. The two QRS complexes are recorded on the electrocardiogram and the nodal P wave is found

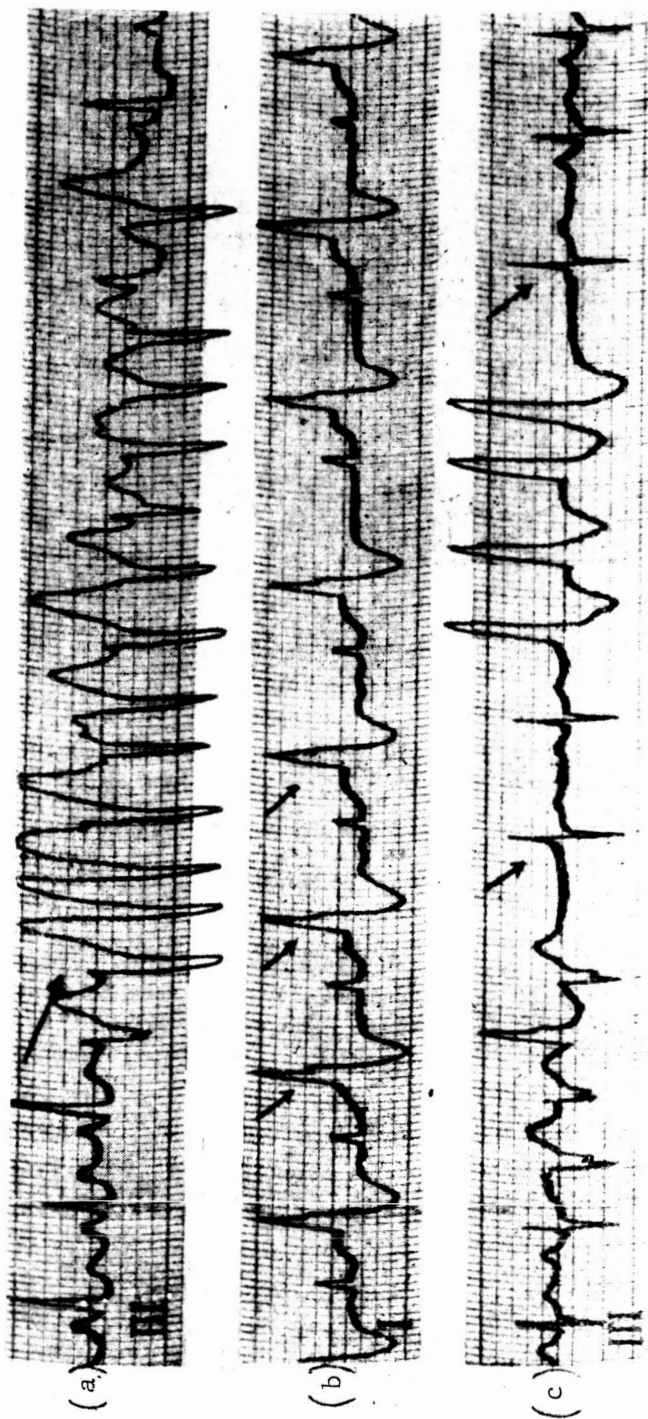


Fig. 35. Extrasystolic arrhythmia. (a) Electrocardiogram of patient B. O., age 31, with following diagnosis: mitral defect with prevalent stenosis. Polytropic ventricular group extrasystoles at the time of digital commissure incision. (b) Electrocardiogram of patient M. V., age 26, with following diagnosis: mitral defect with prevalent stenosis. Ventricular bigeminy against a background of auricular fibrillation. (c) Electrocardiogram of patient M. M., age 39, with following diagnosis: mitral defect with prevalent stenosis. The group ventricular extrasystoles are followed by the formation of escaping systoles from the atrioventricular node (indicated by an arrow).

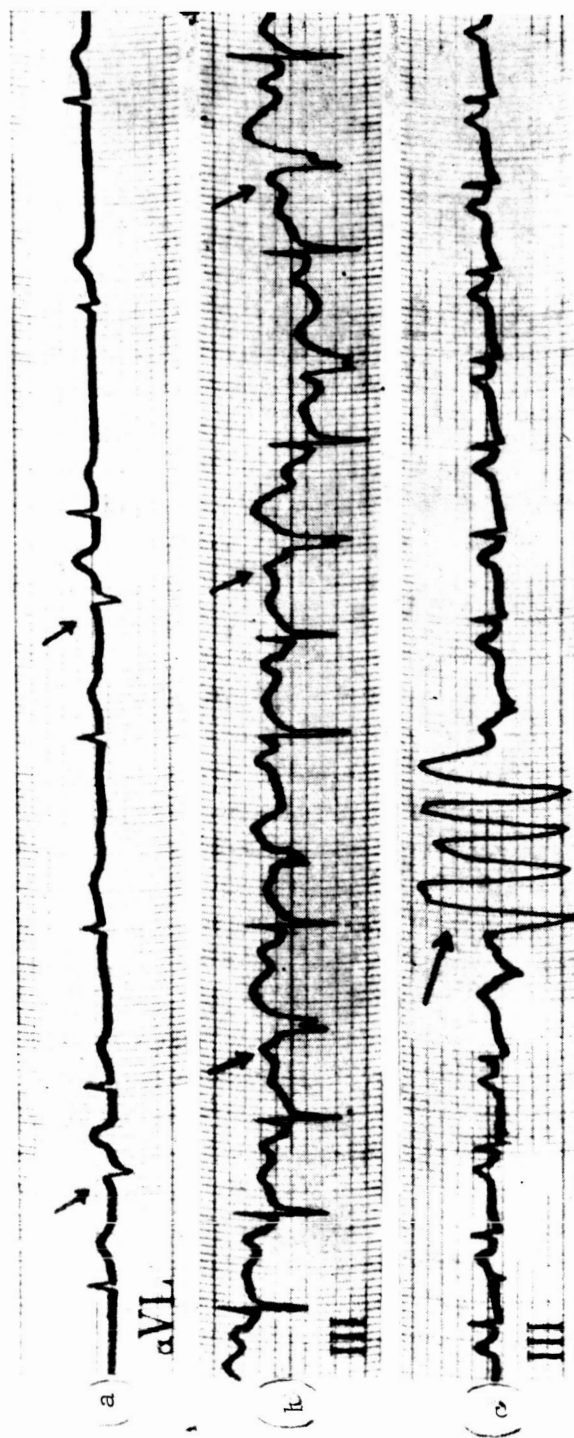


Fig. 36. Extrasystolic arrhythmia. (a) Electrocardiogram of patient S. G., age 30, with following diagnosis: hypertension stage Ib. Two interpolated ventricular extrasystoles. (b) Electrocardiogram of patient M. M., age 29, with following diagnosis: mitral defect with prevalent stenosis. Frequent ventricular extrasystoles three of which are fusion beats. (c) Electrocardiogram of patient B. L., age 22, with following diagnosis: mitral defect with prevalent stenosis. A group of ventricular extrasystoles begins and ends with fusion beats.

in the time interval (usually less than 0.5 sec.) between them.

B. Paroxysmal tachycardia. This form of arrhythmia consists of a sudden paroxysm of a sharply increasing frequency of cardiac contractions which lasts a certain period of time and disappears just as suddenly. The paroxysm may last from several minutes to several days. We have observed a paroxysm over a period of six days in a young man with a congenital heart condition. Katz and Pick (1956) refer to a case of paroxysm that lasted 15 months. The cardiac rhythm during that seizure usually ranged from 150 to 220 beats per minute. Hoffmann and Pomerance (1958) describe a case of tachycardia amounting to 345 beats per minute. Paroxysmal tachycardia is characterized by the /118 actual onset of a very large group of extrasystoles, and this could be proven if it were possible to record the beginning (by the prematurity of the first complex) and the end (by the compensatory pause) of the paroxysm. This is also indicated by the various extrasystolic contractions which frequently precede the onset of tachycardia.

A supraventricular form of paroxysmal tachycardia may be found also in a healthy person; it may be found in members of the same family; and it may even be of a psychogenic origin (Katz and Pick, 1956). Berry (1959) and Montella (1961) describe a case of paroxysm developing with each inspiration. Paroxysmal tachycardia, however, is found to accompany various cardiac diseases, particularly certain heart conditions and cardiosclerosis. The ventricular form is, prognostically, considerably worse and it very seldom occurs in therapeutic practice. We have observed this form in operations connected with various heart conditions and various experimental methods of heart treatment, and noted that it frequently preceded the onset of a ventricular flutter or fibrillation.

The supraventricular form (Fig. 37a) is seldom of sinus origin, more frequently of a precordial and relatively seldom of a nodal origin. We find it more expedient to combine these types into a single group and designate it as a supraventricular form of paroxysmal tachycardia because the only distinctive feature between them is the form of the P-wave, and in the case of a pronounced tachycardia that wave is frequently stratified on the preceding ventricular complexes. In this form the ventricular complexes are unchanged, but they are frequently aberrant because of the functional disturbance of the intraventricular conduction resulting from tachycardia. Paroxysmal tachycardia occasionally deteriorates atrioventricular conduction. Kreisle and Kreisle (1957) describe cases of precordial tachycardia with a 2:1 atrioventricular block, and Schmagranoff and Jick (1957) cite an instance of simultaneous precordial and nodal tachycardia resulting from digitalis intoxication.

In the case of ventricular paroxysmal tachycardia (Fig. 37b), the impulses originate from the ventricles, and all the complexes on the electrocardiogram resemble extrasystoles coming from corresponding ventricles. Hellman and Lind (1956) describe a rare case of digitalis intoxication with the impulse originating from alternating ventricles. The P-waves are usually missing, but they can be observed on rare occasions as a result of

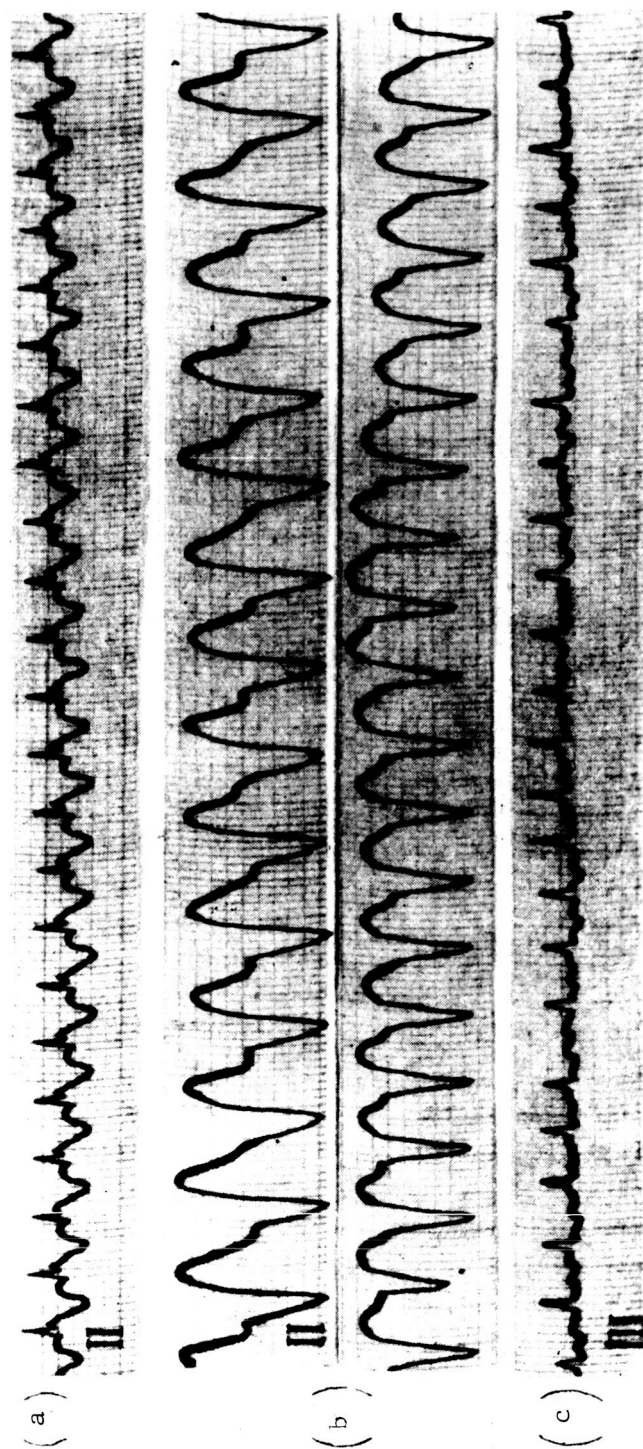


Fig. 37. Paroxysmal tachycardia. (a) Electrocardiogram of patient G. G., age 12, with following diagnosis: congenital vitium cordis (defective interventricular septum?). Supraventricular paroxysmal tachycardia, seizure having lasted six days. (b) Electrocardiogram of patient A. Z., age 25, with following diagnosis: mitral defect with prevalent stenosis. Ventricular paroxysmal tachycardia with a frequency of 300 (upper series) and 400 (lower series) contractions per minute (paroxysm developed during an operation after the digital dilatation of the mitral orifice). (c) Electrocardiogram of patient M. K., age 31, with following diagnosis: mitral defect with prevalent stenosis. Paroxysmal tachycardia against a background of auricular fibrillation.

the retrogressive stimulation of the atrium cordis or, very rarely, as a result of the functioning of the sinus node. In the latter case, the P waves are manifested in the rhythm of sinus automatism.

5. Fibrillation and Flutter

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In this case, some section of the heart fails to produce an adequate contraction and reveals a number of spontaneous and independent tremors of its muscle fibers. No consistency is noted in the electrical phenomena originating in that particular section of the heart, and some part of it is always in an excited state. We find it difficult to agree with the views held by Katz and Pick (1956) according to which flutter or fibrillation is a mechanism whereby the activity of some cardiac chamber is controlled by ectopic impulses and a more frequent rhythm than in paroxysmal tachycardia. But we do agree with these authors that flutter is an intermediate stage between paroxysmal tachycardia and fibrillation, and that there are many intermediate forms between flutter and fibrillation. Fibrillation and flutter are observable in both the atrium cordis and the ventricles.

A. Auricular fibrillation. Of all the other types of arrhythmia, this one is second only to the extrasystole in degree of frequency. It is also known as delirium cordis, arrhythmia absoluta and arrhythmia perpetua. The term fibrillating arrhythmia used in Russian literature had been suggested by G. F. Lang. This type of arrhythmia is observable in mitral stenosis, cardiosclerosis and thyrotoxicosis, and, according to Howard (1960) and G. T. Berdichevskaya (1961), it may also be of a neurogenic origin. It is occasionally possible to restore the sinus rhythm under the effect of the right kind of quinidine therapy. Our observations have revealed that an effective incision of the mitral commissure does not eliminate the auricular fibrillation. A sinus rhythm in the preoperative period was occasionally observed to be followed in the postoperative period by an auricular fibrillation of a transitional nature which rapidly disappeared. That is the paroxysmal form of fibrillating arrhythmia. The prodromes of auricular fibrillation are frequent auricular extrasystoles, seizures of paroxysmal tachycardia and, according to Hundt and Schleimer (1959), a widening of the P wave.

There are different concepts of the mechanism governing the development of fibrillation. Some authors assume the existence of one or numerous ectopic foci which produce impulses at a high frequency. A. M. Segal (1958), for example, finds that unlike the flutter whereby only one heterotopic center of automatism is found in the auricle, fibrillation is accompanied by the activity of numerous stimulatory foci in the atrium cordis. Other authors also point to the importance of the reentry mechanism. The Lewis theory of orbicular motion (1925) has also become popular. This theory is based on the fact that fibrillation is accompanied by a higher excitability of the auricular myocardium which accounts for the shorter duration of the absolute refractory phase, and the conduction is suppressed. The sinus node does not function as a conductor of the cardiac rhythm, and an ectopic impulse originates from the orbicular muscles located around the vena cava

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orifices. That impulse extends in a circular direction along the muscular bundle, and reaches its initial place. As a result of the shortened refractory phase, the excitability of the fiber in this section is restored, and it is capable of being excited again. Such is the origin of constant orbicular motions with a frequency of 200-700 per minute and even higher; they gradually multiply, as the "daughter" circles originate from the "mother" circles.

The theory of orbicular motions is not at present generally accepted, especially after the interesting experimental and clinical projects carried on by Prinzmetal and his colleagues (1951) who, by the use of slow motion pictures demonstrated that no orbicular motion is observable in fibrillation or flutter, and that the ectopic impulse, like the auricular extrasystole or paroxysmal tachycardia, extends in a radiating manner into the auricular myocardium. The authors believe that this arrhythmia comes as a result of deteriorated conduction because the auricular muscles are incapable of conducting such a frequent succession of impulses. The theory of orbicular motion is also opposed by Wiggers (1945) who associates fibrillation with the reentry mechanism.

In auricular fibrillation, the P wave is missing from the electrocardiogram (Fig. 38a) which shows numerous small fluctuations (f) ranging in frequency from 300 to 700 per minute. This undulating iso-electric line is clearly seen in lead III and particularly, in the precordial lead V_1 where

it frequently resembles a flutter (Fig. 38b). The f waves are clearly discernible also in the esophagus leads (V. I. Maslyuk, 1958). P. M. Zlochevskiy (1960) proposes the division of auricular fibrillation into large-wave (over 2 millivolt), medium-wave (0.09-0.19 millivolt), small-wave (up to 0.09 millivolt) and zero (absence of waves) forms. The ventricular complexes, especially the T waves of the various cardiac cycles, are different from one another since the passage of the impulse in the ventricles during each stimulation occurs under dissimilar conditions and, further, the f waves may accidentally get into the individual components of the complex and change them. The complexes appear irregularly, and the observable full arrhythmia of the ventricular contractions occurs because the atrioventricular node is incapable of passing such a large number of impulses, so only a small number of them pass through. The functional condition of the node plays an important part in this problem: the better the condition, the smaller the number of impulses that will pass through the node, the less the work of the ventricles.

There are two forms of fibrillating arrhythmia which are determined by the number of ventricular contractions per minute: bradyarrhythmia (up to 70 contractions) and tachyarrhythmia (more than 80 contractions). There is also a paroxysmal form whereby the auricular fibrillation appears in fits, lasts for some time and disappears. In one of our patients suffering from a mitral vitium cordis, the seizure lasted only several seconds; fibrillation began while the electrocardiogram was being taken, and disappeared by the time it was completed. Auricular fibrillation may occasionally lead to

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a considerable increase in the frequency of ventricular contraction - up to 170-180 per minute. We have frequently observed this in the course of heart surgery, especially before the anesthesia, and attribute it to a unique type of paroxysmal tachycardia (Fig. 37c).

Fibrillating arrhythmia occasionally reveals a complete atrioventricular block (the Frederick symptom). The frequently observed functional block in the ventricles may account for the heterogeneous forms of the ventricular complexes. Only the ventricular extrasystoles can be observed in this type of arrhythmia; there is a possibility of nodal extrasystoles also being formed, but these cannot be detected on the electrocardiogram. A case of myocardial infarction accompanied by a simultaneous auricular fibrillation and ventricular tachycardia has been recorded (Anderson and Rubin, 1958). Of much interest is a case of malignant tachycardia in which precordial extrasystoles developed against a background of sinus rhythm and was followed by a further development of precordial tachycardia which was later replaced by auricular fibrillation, all of which occurred rapidly and recurred periodically (Dewhurst, 1957). We observed a woman patient with an insufficiency of the mitral valve whose paroxysms of auricular fibrillation developed quite often and frequently lasted as long as 9-10 days. The patient (a physician by profession) told us of a very interesting phenomenon: during one of her severe seizures she accidentally made contact with an ordinary household current whereupon the paroxysm immediately stopped (accidental defibrillation). She also told of another case when an accidental contact with an electric wire triggered a new paroxysm.

B. Auricular flutter. The mechanism and etiology of auricular flutter are not different from those of auricular fibrillation. However, there is a quantitative difference between these two forms, and, on the electrocardiogram, to some extent a qualitative difference. Altschule (1954) notes that the symptoms and dynamics of the blood circulation during the flutter are similar to those of tachycardia because the circulation is often accompanied by a frequent ventricular rhythm.

The P-wave is not seen on the electrocardiogram (Fig. 38b) which shows a succession of rapidly rising acuminate (F) waves which occasionally might also be of a two-phase or negative type. The resulting picture is shaped like a saw, and this phenomenon is therefore referred to as the "saw symptom." The waves appear at a frequency of 250-400 per minute, and are clearly outlined in the V_1 , V_2 , III and aVF leads. The ventricular com- /123

plexes are of the same type, and appear regularly but with a lesser rhythm than the F waves; the correlation between them may be 4:1, 3:1 or even 1:1 (Fig. 38c). In the course of our observations we came across an interesting case of an auricular flutter combined with an incomplete atrioventricular block of the II or III type. The patient under consideration, suffering from atherosclerotic cardiosclerosis and 4:1 auricular flutter, periodically revealed a prolapse of the alternate ventricular complex.

C. Ventricular fibrillation. In this case the normal contraction of the ventricles comes to an end, and the uncoordinated stimulation of their

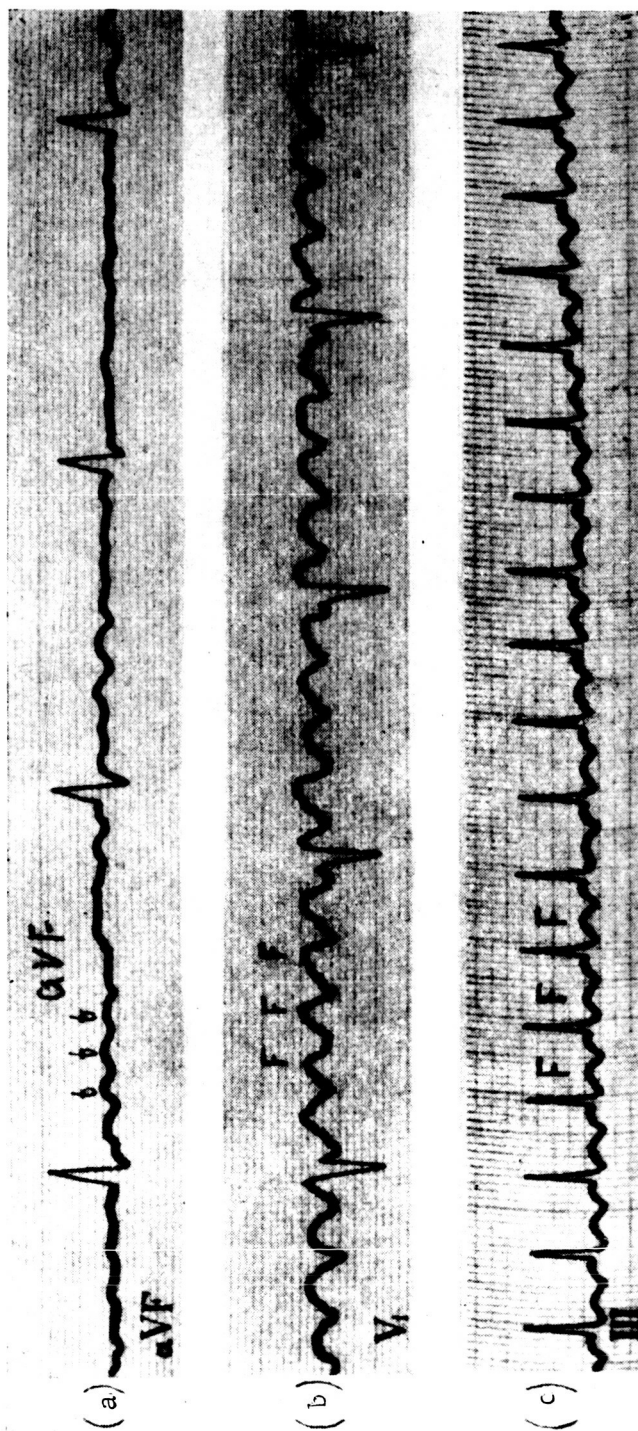


Fig. 38. Auricular fibrillation and flutter. (a) Electrocardiogram of patient K. L., age 57, with following diagnosis: atherosclerotic cardiosclerosis. Auricular fibrillation, well pronounced undulation of iso-electric line (f waves) and irregular rhythm of ventricular contraction. (b) Electrocardiogram of patient P. M., age 37, with following diagnosis: mitral defect with prevalent stenosis. Auricular flutter 5:1, and well pronounced F wave symptom. (c) Electrocardiogram of patient S. A., age 37, with following diagnosis: mitral defect with prevalent stenosis. Auricular flutter 2:1 lasted a very short time.

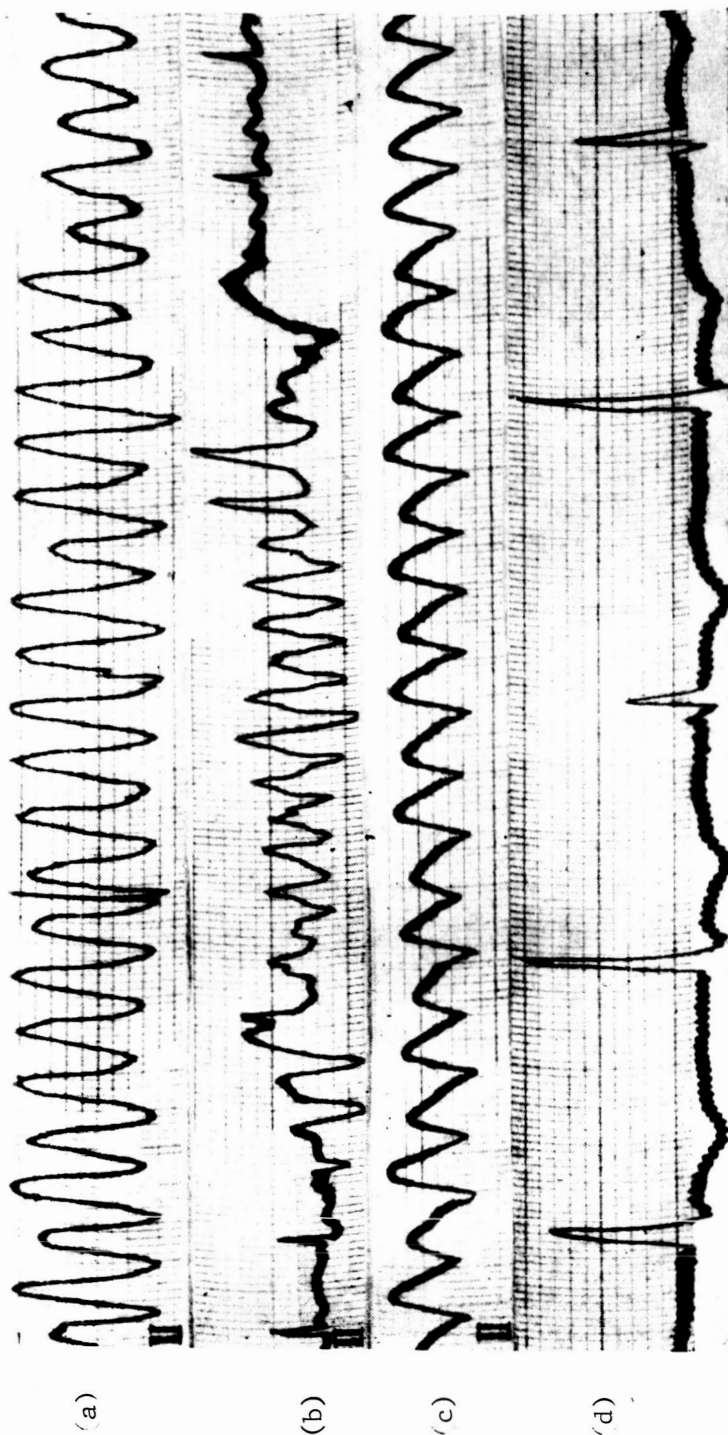


Fig. 39. Ventricular fibrillation and flutter. (a) Electrocardiogram of patient A. Z., age 25, with following diagnosis: mitral defect with prevalent stenosis. Large-wave ventricular fibrillation set in during the rupture of the appendage after the digital dilatation of the mitral opening. (b) Electrocardiogram of patient A. S., age 39, with the following diagnosis: mitral defect with prevalent stenosis. Temporary ventricular fibrillation as the finger was introduced into the left auricle (ventricular extrasystoles noted at beginning and end of fibrillation). (c) Electrocardiogram of patient M. V., age 36, with the following diagnosis: mitral defect with prevalent stenosis. Ventricular flutter precedes cardiac arrest. (d) Electrocardiogram of patient T. A., age 58, with the following diagnosis: atherosclerotic cardiosclerosis. Electric alter-

individual parts begins. This is a very serious phenomenon as it actually results in the arrest of the normal hemodynamics. Ventricular fibrillation is observable during agony, and represents a serious complication in cardiac operations. It is irreversible in most cases, but occasionally it is possible to restore the cardiac rhythm by the use of a defibrillator. Cardiac contractions can easily be restored by the use of various cardioplegic methods to induce fibrillation. The paroxysmal forms of this type of arrhythmia have also been described. In one case we observed a very short and transitional ventricular fibrillation during an operation. It coincided with the incision of the mitral commissure after frequent polytopic ventricular extrasystoles (Fig. 39b). In another case, paroxysm appeared at the time of extubation, lasted 38 seconds and suddenly disappeared.

The electrocardiogram (39a) reveals an irregular succession of undetermined oscillations, dissimilar in form and amplitude and with various bizarre shapes. The P waves are not outlined because they are stratified on these oscillations.

D. Ventricular flutter. It is seldom observed, and represents a serious complication in cardiac operations. The electrocardiogram (Fig. 39c) reveals a regular succession of high-frequency oscillations which are entirely different from the ordinary ventricular complexes.

6. Electric Alternation

An alternating pulse which is very seldom accompanied by an electric alternation can occasionally be found in a clinical examination, particularly one involving the use of an ordinary pulse sphygmograph. Electric alternation is observable in a pronounced paroxysmal tachycardia, but it can serve as an index of severe myocardial infarction only in the case of a normal cardiac rhythm or bradycardia.

The explanation for this electrocardiographic phenomenon (Fig. 39d) is that the amplitude and form of the ventricular complex change. The amplitude of the R-wave is reduced and the wave becomes indented at certain intervals, usually after each contraction. The P waves in such cases appear as usual.

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Some authors associate the electric alternation with the changing position of the heart, especially when there is fluid in the pericardial cavity. Others assume the presence of an elongated phase in the individual parts of the myocardium with the result that these sections can be penetrated only by every other impulse which finds them outside the refractory phase.

7. Hypertrophy

Hypertrophy may be isolated in one particular section of the heart, or combined in several or symmetrical sections simultaneously. The development of hypertrophy changes the normal depolarization and repolarization processes in a given section of the heart. In addition, it disrupts the normal cor-

relation between the muscular masses of the two symmetrical sections of the heart resulting in a change of its position.

A. Auricular hypertrophy. Observable in different pathological conditions. A typical hypertrophy of the atrium sinistrum is produced primarily by mitral stenosis, and the atrium dextrum by a chronic pulmonary heart disease. It is difficult to detect hypertrophy on an electrocardiogram because only the depolarization phase of the auricular stimulation is clearly reflected on the curve.

The hypertrophy of the atrium sinistrum (Fig. 40) is accompanied primarily by an increasing duration of the P wave and the appearance of typical double-peaked mitral P waves in the standard leads, particularly in I and II. In the aVR lead the P wave is negative and in the aVL and aVF leads it is positive but widened and acuminate. A double-peaked P wave with a relatively more pronounced vertex is noted in the left precordial positions. A two-phase P wave with a somewhat acuminate first positive phase and a slower and wider second negative phase are frequently noted in the right precordial positions.

The hypertrophy of the atrium dextrum (Fig. 41) is accompanied primarily by an increasing P wave amplitude and the appearance of typical acuminate pulmonary P waves in the standard leads, particularly II and III. The P wave is negative in the aVR lead, iso-electrical or negative in the aVL lead and positive in the aVF lead. In the $V_{1,2}$ it is positive,

enlarged and double-peaked (frequently with a pronounced first vertex) or diphasic (with a pronounced first wide positive phase and second acuminate negative phase). In the left pectoral positions the P wave is frequently positive and somewhat enlarged.

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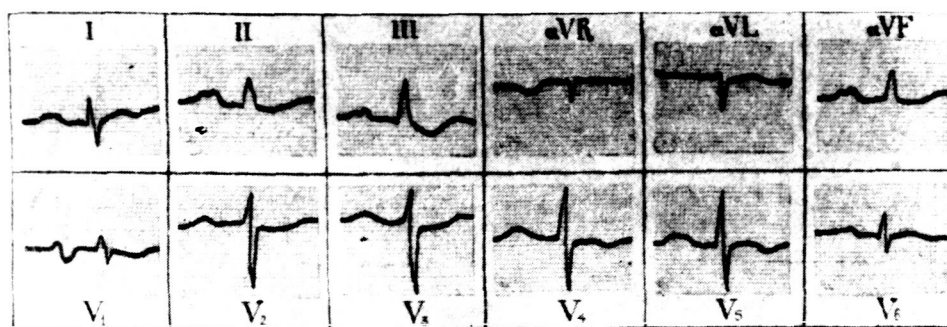


Fig. 40. Hypertrophy of the atrium sinistrum. Electrocardiogram of patient Kh. A., age 27, with following diagnosis: mitral stenosis. Wide mitral P waves clearly outlined in I, II, III, aVR, aVF and V_5 leads; a two-phase

P wave with a wider and slower second negative phase is found in the V_1 leads.

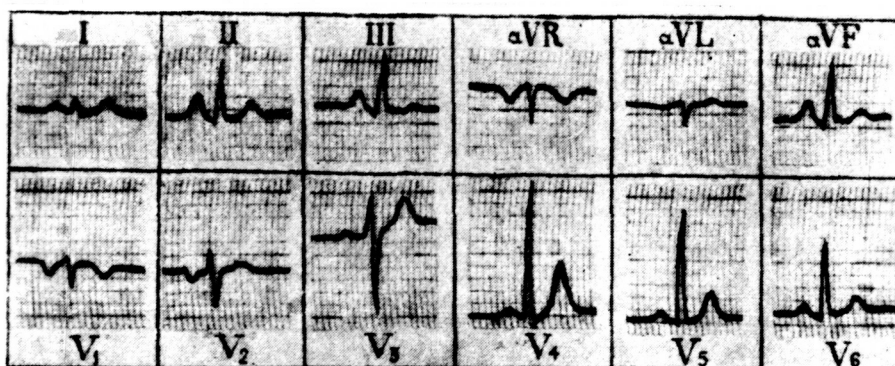


Fig. 41. Hypertrophy of the atrium dextrum. Electrocardiogram of patient K. M., age 29, with following diagnosis: mitral defect with prevalent stenosis, tricuspid valve insufficiency. Pulmonary P waves clearly outlined in II, III and aVF leads. The P wave is negative in V_1 and two-phased in V_2 .

As Kahn et al. (1960) point out, the Macruz index is of relative or auxiliary importance in auricular hypertrophy. It is more than 1.5 in the hypertrophy of the atrium sinistrum, and less than 1.0 in the hypertrophy of the atrium dextrum.

A combination of all the above-described changes is observable in the hypertrophy of both auricles.

B. Ventricular hypertrophy. Left ventricular hypertrophy is observable in cases of aortic damage, insufficiency of the mitral valve, hypertension, cardiosclerosis, etc. Hypertrophy of the right ventricle occurs in mitral stenosis, cor pulmonale, tetralogy of Fallot, stenosis of the pulmonary artery, etc. A combined hypertrophy of both ventricles is a fairly frequent phenomenon, particularly in mitral damage, mitral-aortic defect, patent ductus arteriosus, etc. /128

Left ventricular hypertrophy (Fig. 42) reveals wide and high QRS complexes, and the rising QRS amplitude is an early symptom of left ventricular hypertrophy (Grubschmidt and Sokolow, 1957). A left type of electrocardiogram appears in the standard leads with a high R wave and, frequently, with the RS-T segment displaced downward with some upward convexity, and a negative T wave in lead I. The electrical axis is turned to the left, and the Lewis index is more than +20 mm. If the heart is in a horizontal position, the aVR lead reveals a ventricular complex of the rS or QS type, the T wave is usually positive, a qR-shaped complex with a negative T wave is recorded in the aVL lead, the RS-T segment is a little lower than the iso-electric line, and the ventricular complex in the aVF lead is usually shaped like an rS with a positive T wave. In the less frequently observable vertical position of the heart, rS or QS-type complexes are usually recorded in the aVR and aVI lead, the T wave is positive, and a qR-type complex with a downward displaced RS-T segment and negative T wave are observable in the aVF lead.

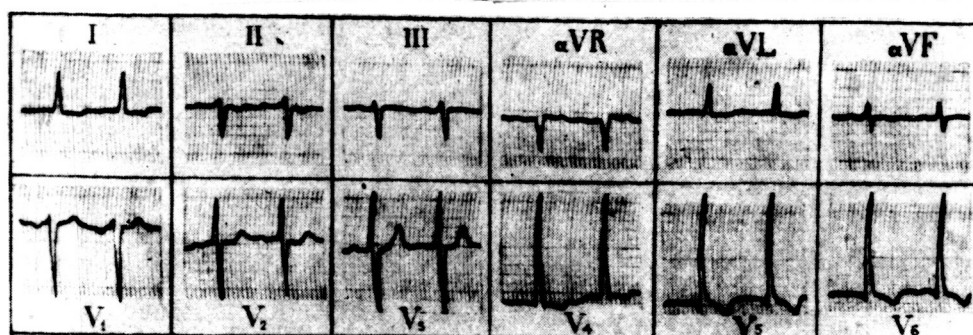


Fig. 42. Hypertrophy and the strain of the left ventricle. Electrocardiogram of patient S. B., age 49, with following diagnosis: hypertensive disease, stage IIb. Electrical picture of hypertrophy and strain of the left ventricle is well pronounced.

In left ventricular hypertrophy, the left precordial leads reveal typical qR or qRs-type complexes with a high amplitude of the R wave; the RS-T segment is displaced downward with some upward convexity, and the T wave is frequently negative. In these positions, the origin of the internal deviation lasts a little longer; it is usually more than 0.045 sec. The ventricular complexes in the right precordial leads have deep S waves, occasionally even a QS form, the RS-T segment is displaced upward and the T wave is positive. The time of the origin of the internal deviation is unchanged. When the heart is in a normal position the transitional forms of the ventricular complex are observable in the V_3 lead. As the heart is

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turned counterclockwise around a longitudinal axis, the transitional complexes are seen in the right positions, and when it is turned clockwise, such complexes are found in the left positions.

Left ventricular hypertrophy is frequently accompanied by an incomplete or complete bundle branch block.

The Sokolow and Lyon (1949) criteria for left ventricular hypertrophy are valuable, and we list them below:

$$R_1 + S_{111} = 25 \text{ mm}; \quad R_{aVL} \geq 11 \text{ mm};$$

$$R_{V_5} \text{ or } V_6 \geq 26 \text{ mm}; \quad R_{V_5} + S_{V_1} \geq 35 \text{ mm};$$

The time of the origin of the internal deviation

$$\text{in lead } V_6 \geq 0.06 \text{ sec.};$$

The displacement of the RS-T segment in the following

$$\text{leads: } V_4 \text{ and } V_5 \text{ or } V_6 \text{ and } aVL \text{ or } aVF \geq 0.5 \text{ mm.}$$

It should be pointed out that these criteria are found only in acute types of left ventricular hypertrophy.

The extent of the widening and increasing amplitude of the QRS complexes in right ventricular hypertrophy (Fig. 43) is not as great as in left ventricular hypertrophy. The standard leads reveal a right type of electrocardiogram with a deep S wave in lead I and a high R wave in Lead III where one can observe a downward displacement of the RS-T segment with some upward convexity and a negative T wave. A right turn of the electrical axis is noted, and the Lewis index is less than -15 mm. The verticle position of the heart reveals a Qr or QR-type ventricular complex in the aVR lead, the T wave is usually negative and an rS shaped complex with a positive T wave is recorded in the aVL lead; the ventricular complex in the aVF lead has the form of qR or qRs with a positive (or negative) T wave. In the horizontal position of the heart, which is observable considerably less often, a qR or QR-type complex is usually recorded in the aVR lead, T is negative, the qR or QR form with a positive T wave appear in aVL, and a ventricular complex shaped like R or RS with a negative T wave in the aVF lead.

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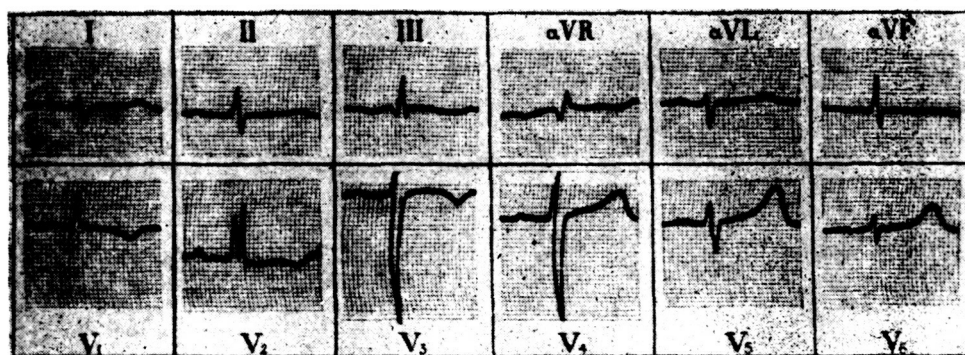


Fig. 43. Hypertrophy of the right ventricle. Electrocardiogram of patient M. M., age 39, with following diagnosis: mitral defect with prevalent stenosis. Hypertrophy of the right ventricle, partial right bundle branch block; systolic as well as diastolic overload of the right ventricle (according to the Cabrera and Monroy criterion).

In right ventricular hypertrophy, the right precordial leads reveal complexes with a predominate R wave, the RS-T segment is displaced downward with the convexity facing upward, and the T wave is usually negative or two-phased. In this position, the origin of the internal deviation takes more time; it is longer than 0.03 sec. In the left precordial leads the ventricular complexes have deep S waves which are usually shaped like rS or RS, and the T wave is positive; the origin of the internal deviation remains unchanged.

An incomplete and complete block of the right branch of the bundle of

His is fairly frequently observed in right ventricular hypertrophy. Goodwin and Abdin (1959) determine the presence and extent of right ventricular hypertrophy on the basis of the V₄R and VR lead. Roman, Walsh, and Massie (1961) make use of the R/S coefficient at the chest leads and the extent of axis turn to the right for the same purpose. The most detailed electrocardiographic criteria of right ventricular hypertrophy are cited by Sokolow and Lyon (1949) (the criteria listed below are observable in pronounced degrees of right ventricular hypertrophy):

$$\begin{aligned}
 Rv_1 &\geq 7 \text{ mm (58\% of the cases);} & /131 \\
 Sv_1 &\leq 2 \text{ mm (50\% of the cases);} \\
 Sv_6 &\geq 7 \text{ mm (50\% of the cases);} \\
 R_v + Sv_5 &\geq 10.5 \text{ mm (72\% of the cases);} \\
 Rv_5 &\leq 4 \text{ mm (35\% of the cases);} \\
 R/Sv_5 &\leq 1 \text{ mm (32\% of the cases);} \\
 Ra_{VR} &\geq 5 \text{ mm (30\% of the cases);} \\
 R/Sv_5/R/Sv_1 &\leq 0.4 \text{ (25\% of the cases);} \\
 R/Sv_2 &> 1 \text{ (22\% of the cases);} \\
 &\text{the time of the origin of the internal deviation} \\
 &\text{at lead } V_{11} \leq 0.04 \text{ sec. (70\% of the cases);} \\
 &\text{negative } Tv_1 \text{ with } Rv_1 \geq 5 \text{ mm (42\% of the cases).}
 \end{aligned}$$

It should be pointed out that it is relatively more difficult to diagnose the hypertrophy of the right ventricle than of the left one. This problem is becoming increasingly important in view of the growing scope of surgical treatment applied in cases of mitral stenosis and a number of congenital heart conditions. Our observations show that the aVR lead of the EKG, which reveals a high late R wave, and particularly the right precordial leads, including V₁, which may record three types of a ventricular complexes (R, qR, rS

or RS), are important for the diagnosis of right ventricular hypertrophy in the case of mitral stenosis (we do not share the belief of Camerini, Goodwin and Zoob (1956) and Morton (1960) that the V₃R and V₄R leads supply more important data). It has been found that the presence of ventricular complexes in the V₁ lead with a prevalent R wave, which is not connected with the late intracavitary R wave observable in this particular case of hypertrophy, is the most effective indicator of right ventricular hypertrophy (Emslie-Smith, 1956). The qR-shaped complexes are indicative of a pronounced hyper-

trophy of the right ventricle, and the rS or RS-type complexes are found mostly when the left ventricle is also affected by hypertrophy at the same time. We have observed a definite and direct relationship between the degree of mitral stenosis and the extent of the mentioned criteria.

There is some difficulty in diagnosing a combined hypertrophy of the ventricles. In the case of a prevalent left ventricular hypertrophy the hypertrophy of the right ventricle may not even be detected as it tends to complicate the following normal correlation:

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left ventricle mass
right ventricle mass

The prevalence of right ventricular hypertrophy makes the detection of left ventricular hypertrophy doubtful. Te-Chuan Chu et al. (1960) found that the available criteria are inadequate for differentiating left ventricular hypertrophy from a combined hypertrophy of both ventricles. We assume that a detailed analysis of all the 12 leads, especially the precordial leads, and the use of all the described criteria would also make a diagnosis of combined ventricular hypertrophy possible. It is possible to distinguish the following most common combinations of electrocardiographic indicators of combined ventricular hypertrophy (taking into account the turns of the heart around the three basic axes).

1. All the criteria of a pronounced left ventricular hypertrophy are found in the precordial leads and the amplified leads of the extremities. The QRS voltage in the standard leads is not increased, and there is some indication of right ventricular hypertrophy. Such a picture is observable in the case of a prevalent left ventricular hypertrophy.
2. Criteria of left ventricular hypertrophy are found in the left precordial leads and in the aVL and aVF leads, while the S wave in the precordial leads is shallow, and the R wave is somewhat enlarged and a small late R wave appears in the aVR lead. Criteria of right ventricular hypertrophy are found in the standard leads. Such a picture is observable in a similarly pronounced hypertrophy of both ventricles.
3. All the indicators of right ventricular hypertrophy are found in the right precordial leads and the six limb leads. In the left precordial leads the R wave is of the usual or somewhat reduced magnitude, and the S wave is either shallow or missing. Such a picture is observable in a prevalent hypertrophy of the right ventricle.
4. Indefinite combinations of indicators in various leads complicate the diagnosis of a prevalent hypertrophy of a particular ventricle. In some cases, all the described criteria of ventricular hypertrophy may not be shown on the electrocardiogram if the hypertrophy is equally pronounced in the left and right ventricles. This is apparently due to the neutralization of the opposite electrical forces.

C. Regression of cardiac hypertrophy. The development of hypertrophy following the elimination of the cause behind the hyperfunction of a given section of the heart is a matter of great interest. The experimental aspects of the hyperfunctions are thoroughly discussed in F. Z. Myerson's original studies (1960). Our own clinical observations (Z. L. Dolabchyan and N. G. Tatinyan, 1962) showed that a surgical correction of a vitium cordis is soon followed by a reverse development of hypertrophy. Thus an effective mitral commissure incision in conditions of the new hemodynamics is followed (Table 4) by a reduction or disappearance of the hyperfunction of the right ventricle and the left atrium, and the electrocardiogram reveals a gradual discharge of the right ventricle (a reduction of the S wave in lead I and the R wave in the aVR lead; a reduction of the R wave and the appearance or enlargement of the S wave in the $V_{1,2}$ leads and a reduction of the S wave in the V_5 leads) and the left atrium (an improvement and normalization of the amplitude, widths and form of the P wave in the I, II, aVR, aVF and $V_{1,2}$ leads in the case of a sinus rhythm). There is also an improvement in the nutrition of the right section (an improvement and normalization of the RS-T segment and T wave in the $V_{1,2}$ leads.) The relief of the right ventricle is accompanied by an increasing load on the left ventricle (an increase of the R wave in lead I; the appearance of an S wave in lead III; a reduced amplitude of the R wave and increased amplitude of the S wave in the $V_{1,2}$ leads; an increasing R wave; a reduced RS-T segment and lower amplitude of the T-wave in the III, aVL and $V_{5,6}$ leads.) This picture lasts several months (Fig. 44). As a result of such a deterioration of the nutrition of the myocardium in the left ventricular region (and occasionally also in the area of the right ventricle) over a lengthy period of time, a parallel between the good condition of the patient and the picture of electrical cardiac activity cannot be observed. It should be assumed that surgical correction improves the hemodynamics and brings the nutrition of all the organs and tissues of the organism closer to the norm, but the heart does not immediately adapt itself to the new conditions of the intracardiac hemodynamics. This applies, first of all, to the left ventricle whose function before the operation was inadequate. An increase in the cardiac beat volume after the operation puts a heavy load on the untrained muscles of the left ventricle.

If in the case of a mitral-aortic commissure incision the dynamics of the reverse development in the right ventricle occurs in the same general direction as after an isolated mitral commissure incision, the picture observable in the case of the left ventricle is entirely different (Table 4): the amplitude of the R wave in lead I is reduced, a gradual reduction of the R wave amplitude is observable in the left precordial leads, and the amplitude of the R wave in the right precordial leads gradually becomes smaller. These indicators point to the dynamics of the reverse development

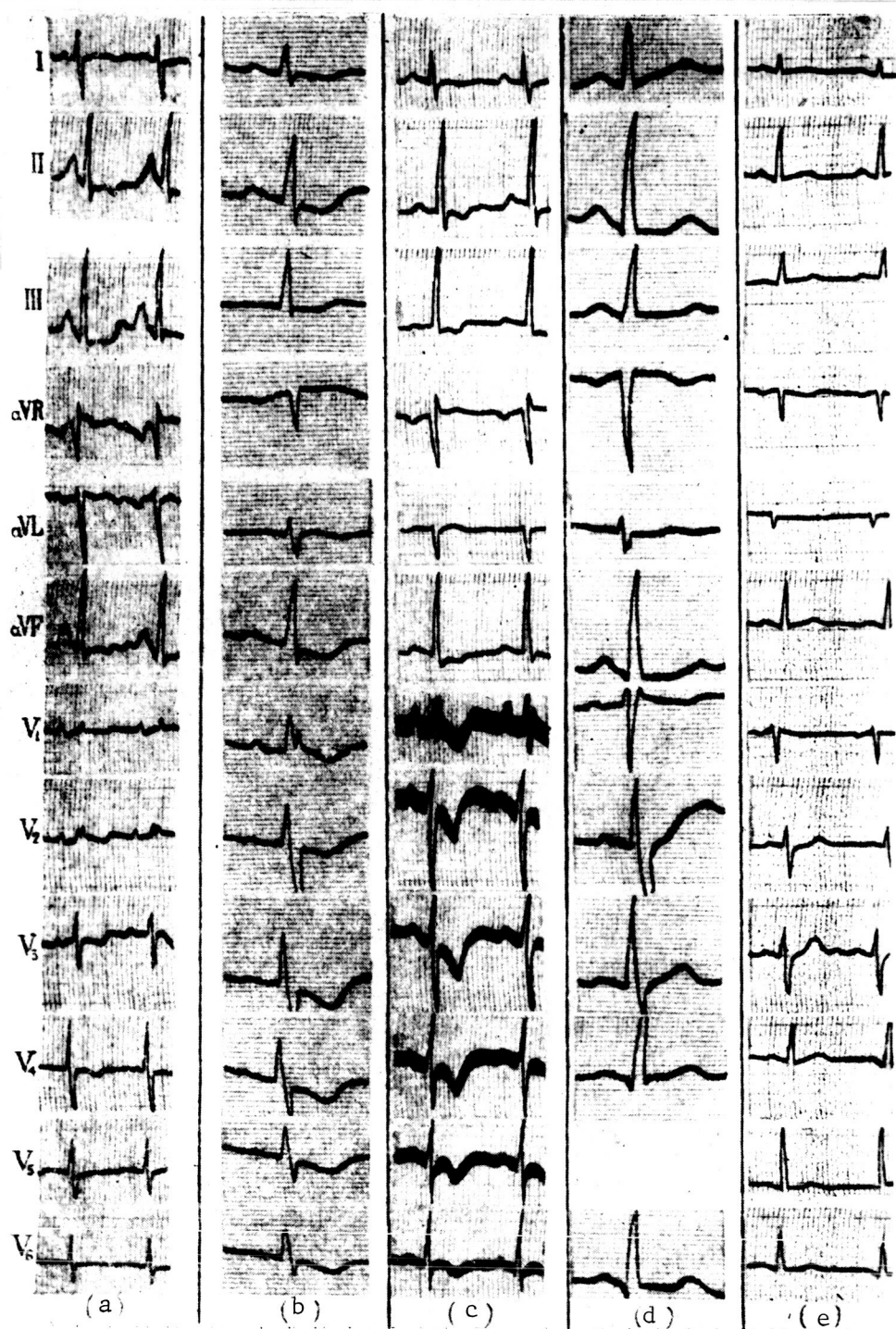


Fig. 44. Regression of cardiac hypertrophy. Electrocardiogram of patient S.V., age 34, with following diagnosis: mitral defect with prevalent stenosis. Curves: (a) before the operation, (b) three weeks after, (c) 1.5 months after, (d) 4.5 months after, and (e) one year after the mitral incision. The dynamics of electrocardiographic data point to gradual relief of the left auricle and of the right ventricle; the strain on the left ventricle practically disappears after approximately one year.

of hypertrophy in the left ventricular region.

D. Ventricular strain. This electrocardiographic concept implies a number of electrocardiographic criteria. A comparison of the strain to the hypertrophy is made from a purely electrophysiological point of view. The term was introduced into electrocardiography as far back as 1929 by Barnes and Whitten (1929), but the strain concept was developed primarily by other authors. Goldberger (1954) sees the following difference between ventricular strain and hypertrophy: hypertrophy is the result of a big overload on the ventricle lasting a long time, whereas strain is produced by an overload which, though heavy, lasts a short time and is insufficient for the development of hypertrophy. Ventricular strain may be produced even in a healthy heart by an acute disruption of the appropriate department of blood circulation.

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Ventricular strain is not reflected on the electrocardiogram (Fig. 42, 43) by an increase in the QRS voltage or retardation of the intra-ventricular conduction, which are characteristic of hypertrophy, but in a change of the other parts of the ventricular complex: the RS-T segment is displaced downward with its convexity upward, and the T wave becomes symmetrical and negative. In the case of left ventricular strain, these changes are observable in the left precordial leads, in lead I and, depending on the position of the heart, in the aVL or aVF lead, while in a right ventricular strain they are found in the right precordial leads and in the III and aVF leads. There are systolic and diastolic overloads, according to Cabrera and Monroy (1952): the systolic overload leads to hypertrophy, and the diastolic overload leads to a dilatation of the given ventricle. A systolic overload of the left ventricle reveals a downward displacement of the RS-T segment and an inversion of the T waves in the left precordial leads; in a diastolic overload the amplitude of the R wave is increased and the origin of the internal deviations in these leads takes a longer time. A systolic overload of the right ventricle is accompanied by an increase in the R wave and the presence of T waves in the right precordial leads; a systolic overload of the same ventricle is followed by the appearance of different versions of the sRR'-type complex and a longer duration of the internal deviation in these same leads. P. L. Gladyshev (1962) finds that the retardation of the internal deviation is due primarily to dilatation, and the increase in the amplitude of the R wave is due to the hypertrophy of the appropriate ventricle.

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We assume that it is also possible to detect an auricular overstrain on the electrocardiogram since this phenomenon is apparently accompanied by a considerable increase in the P wave and displacement of the P-Ta segment. We made this assumption on the basis of our observations of mitral and aortic commissure incisions.

Some authors associate the above described so-called electrocardiographic functional changes occasioned by ventricular strain with changes in the pH of tissue surrounding the strained muscular fibers, others associate them with myocardial anoxia or ischemia, and still others attach some significance to hypokalemia (according to Goldberger 1954). It should be pointed out

Table 4

Changing EKG wave amplitudes (in mm) after the operation on patients suffering from mitral stenosis and mitral-aortic stenosis

EKG waves	Mitral stenosis			Mitral-aortic stenosis		
	Preoperative	Postoperative		Preoperative	Postoperative	
		1 month	3 months		1 month	3 months
P ₁	2.4±1.281	1.5±0.911	1.4±0.848	1.6±0.843	1.6±0.728	1.3±0.742
Pv ₁	1.8±1.706	2±1.414	1.5±0.911	1.6±0.911	0.9±0.583	2±0.618
Qv ₁	1.2±0.648	2±2.074	2.6±2.668	0.61±0.990	0.72±0.652	1.2±0.700
R ₁	3.3±1.850	5±3.609	5.6±4.135	4±2.098	5±1.414	2.1±1.726
Fv ₁	5.3±4.350	6.1±3.976	4.1±3.871	5.1±2.577	4±1.871	3±2.098
Fv ₆	9.5±5.254	9.1±6.299	12.5±5.357	11.1±5.173	10±2.943	10.2±3.647
S ₁	3.8±3.154	3.5±3.421	2.6±2.147	3.1±1.855	2.1±1.881	2.1±0.990
Sv ₁	4.4±3.231	4.6±4.284	6±5.099	5.6±2.309	5.5±3.975	4.2±1.980
Sv ₆	4±3.479	2.9±1.908	1.5±0.866	5.2±2.707	2.3±2.098	2.5±1.349
T ₁	1.8±1.145	2.1±1.723	2±1.225	2.3±0.943	1.4±0.83	1.2±0.812
Tv ₁	1.5±0.634	2.4±2.040	1.8±1.221	1.8±0.800	1.6±0.735	1.2±0.700
Tv ₆	3.4±1.876	2.1±1.421	2.3±1.364	4.3±1.300	1.4±0.83	1.7±0.985

that a number of authors object to the term and concept of strain. L. I. Fogelson (1957) objects to this method of treating the problem on general principles, Sodi-Pallares (1956) never mentions the term strain at all, and Mayers (1956) believes that this term is unacceptable because it implies a mechanical phenomenon. Lenegre, Carouso and Chevalier (1954) consider strain to be the initial stage in the development of hypertrophy. Jouve, Senet and Pierron (1954) see no difference between strain and hypertrophy, and V. Ye. Nezlin and S. Ye. Karpay (1959) juxtapose the term strain with the term overload, considering the former as an inadequate load for the myocardium. We are inclined to believe that even if an objection to the term strain is justifiable, there must be something in this electrocardiographic category /137

that distinguishes it from hypertrophy. To an electrocardiologist strain is a more dynamic concept connected with a definite hemodynamic factor (rising blood pressure, for example) which produces strenuous conditions for the ventricular myocardium, whereas hypertrophy is more of a static concept (compared to strain) which includes also the moment of strain. For example, a good hypotensive affect in a patient suffering from hypertension is reflected on the electrocardiogram, first of all, by an improvement or normalization of the RS-T segment and T wave indicators. The reduction of the R wave amplitude occurs relatively later. Or, in addition to the reduction of the hypertension in the lesser blood circulation, an effective mitral commissure incision is followed by an increasing amplitude of the T wave and normalization of the RS-T segment in the right precordial leads, and the R wave is decreased in the later periods, but in the first month it even grows somewhat larger. It should be assumed that strain is reflected on an electrocardiogram primarily in the displacement of the RS-T segment and the depression of the T wave, while hypertrophy is manifested in the increasing amplitude of the QRS complex and retardation of the internal deviation. With such an approach, it is difficult to differentiate between the systolic and diastolic overload, as the same changes may be observed in these two types of strain.

We believe that the above described idea of the electrophysiological phenomenon of strain enriches our knowledge in the field of electrocardiology.

8. Electrolytes of the Blood

Among the various electrolytes of the blood that play a major part in the mechanism of electrical phenomena occurring during the cardiac contraction are the potassium and calcium ions.

A. Potassium. The gradient of the intra- and extracellular content of potassium in the blood plays such an important part in the stimulation of the heart that certain authors even believe it possible to determine the quantity of potassium on the basis of the characteristic electrocardiographic changes observable in hypo- or hyperkalemia (abnormal deficiency or excess of potassium in the blood). The potassium content in the blood serum may to some extent be used as a basis for clinical studies.

Hypokalemia is observable in certain cases of chronic nephritis, diabetic acidosis, chronic diarrhea, the wrong use of cortisone therapy, etc. It is reflected on the electrocardiogram in the repolarization phase and decreasing T wave; the more pronounced the hypokalemia, the greater the decrease in the T wave amplitude. The other components of the electrocardiogram remain unchanged, although a pronounced U wave and some displacement of the RS-T segment are frequently observed. Extrasystoles are frequent. Sanghvi and Misra (1958) describe that WPW syndrome of hypokalemia.

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Hyperkalemia is observed in certain cases of acute or chronic nephritis accompanied by azotemia, in Addison's disease, etc. On the electrocardiogram the P wave appears somewhat shortened and low, the Q-T interval is considerably shortened, the RS-T segment is somewhat displaced downward, and the T wave reveals a typical picture, symmetrical and high with a pointed vertex. The studies made by V. S. Sal'manovich (1954-1959) and other authors show that the degree of such changes is determined by the level of the hypokalemia. The latter phenomenon may involve various disruptions of conductivity in every stage of the conduction system. In such cases, the existing extrasystoles disappear.

B. Calcium. The concentration of the calcium ions plays an important part in the depolarization of the cardiac muscle fibers.

Hypocalcemia is observable in spasmophilia, tetany, nephritis, etc. In such cases, the QRS complex on the electrocardiogram shows no change, but the Q-T interval is lengthened by the widening of the characteristic horizontal RS-T segment. The extent of the Q-T interval increase is directly proportional to the level of the hypocalcemia (Bekhtel et al., 1957). The T wave is somewhat decreased and occasionally negative, and a combined TU wave comes into view (Hegglin, 1957).

Hypercalcemia is more frequently observable in hyperparathyroidism. In this case the Q-T interval is greatly shortened as is, particularly, the repolarization phase which sets in very early and may even be missing from the RS-T segment. The rapid development of hypercalcemia results in an extension of the cardiac cycle, primarily through the T-P interval (Harris and Levin, 1937), a disruption of the atrioventricular conduction, a ventricular extrasystole and even fibrillation. Hyperparathyroidism may develop the Wenkebach-Samoylov phenomenon (Krum and Till, 1960).

9. Specific Medicines

A. Digitalis and similar cardiac glucosides. Digitalis produces various disruptions in the rhythm of cardiac activity as well as changes of a morphological nature. These phenomena, occurring during the treatment, or in digitalis toxicity, are well known in literature and their clinical importance has been established. The mechanism of the mentioned changes is connected with the direct effect of digitalis on the myocardium and the increasing tonus of the vagus nerve. The direct effect of digitalis on the coronary blood circulation, in this connection, cannot be ruled out. It should be pointed out also that the therapeutic or toxic effect of digitalis is associated with its effect on the potassium ion exchange (Pick, 1957). /139

The auricular complex on the electrocardiogram is usually unchanged. There is an occasional decrease in the voltage of the P wave. Very characteristic are the shortening of the Q-T interval with the changing RS-T segment and T wave. The RS-T segment is displaced downward, at first in a slanted downward direction and at the end it rises sharply to the iso-

electric line. The T wave voltage is reduced, and may become active in the case of a pronounced intoxication. A number of varied types of arrhythmia come into being: sinus arrhythmia, the migration of the cardiac pacemaker, various extrasystoles, nodal rhythm, various types of an atrio-ventricular block, etc. Auricular fibrillation with a tachysystole is followed by a decreasing number of cardiac contractions, although Bacaner (1958) describes a paradoxical phenomenon whereby the frequency of ventricular contractions is sharply increased under the effect of digitalis intoxication.

B. Quinidine. If digitalis increases myocardial stimulation and suppresses conduction, quinidine extends the duration of the refractory phase thereby reducing myocardial stimulation. The QRS complex on the electrocardiogram remains unchanged, but the Q-T interval is lengthened through the widening of the T wave. Cheng, et al. (1956) believe that the T wave does not become wider but that its actual change involves also the U wave. The T wave amplitude is frequently reduced. Negative T waves are seldom possible. Proper quinidine therapy changes auricular fibrillation to flutter which is then replaced by a sinus rhythm. A change in the dosage may reveal various complications in the form of extrasystoles, bundle branch blocks, or atrioventricular blocks.

In actual practice, both digitalis and quinidine are used in the treatment of auricular fibrillation even though these preparations have different action mechanisms. Digitalis improves the cardiac contraction (intensifying and shortening the systole and lengthening the diastole) and deteriorates the conduction, but on the other hand it raises the already high level of myocardial stimulation observable in pathology. In view of this, digitalis should be used only in the tachyarrhythmic form of fibrillating arrhythmia with a view to reducing the number of ventricular contractions and improving the blood circulation as a whole. Goldberger (1954) writes that digitalis used in cases of auricular flutter increases the wave frequency and changes the flutter to fibrillation. If the use of digitalis is discontinued in this phase, the fibrillation disappears and the sinus rhythm is restored. This effect appears to us to be paradoxical, and the author himself cannot explain it.

Quinidine is more effective in the treatment of auricular fibrillation /140 as it suppresses two important links of the fibrillation mechanism - atrio-ventricular conduction and cardiac stimulation. It is frequently possible to restore the normal sinus rhythm by the proper use of quinidine therapy.

10. Cardiac Surgery

Constant electrocardiographic observation is a very important element in the surgical treatment of the heart. Such a study may establish certain patterns in the various changes of the cardiac functions produced by the correction of various defects. The interpretation of such patterns will enrich the electrocardiological science. Moreover, continuous electrocardio-

graphic observation in the course of surgery is a necessary element in the work of the cardiac team, and experience shows that it is occasionally the only guide to the solution of urgent problems. If a continuous electrocardiographic recording is required for scientific purposes, an observation of the cardiac condition on an oscilloscope screen is quite adequate for practical purposes.

Different surgeries affect the electrical activity of the heart in different ways. Surgeries on blood vessels (in cases of tetralogy of Fallot (the use of anastomosis) and patent ductus arteriosus) are accompanied by small changes. These consist primarily of a more frequent rhythm of cardiac activity, extrasystoles and some deterioration in the supply to the myocardium. Very big changes are observable during mitral or aortal commissure incisions. These phenomena are described in various sections of this chapter. On the whole, it may be said that the following changes are observable at various stages of these operations (Z. L. Dolabchyan, M. A. Yesayan and N. G. Tatinyan, 1962).

1) Almost all of the patients reveal a faster rhythm of cardiac activity even before the anesthesia. Occasional or frequent extrasystoles are not uncommon and paroxysmal tachycardia occurs less often.

2) Intubation is frequently accompanied by some deterioration in the supply to the myocardium. Turning to a side position does not particularly affect the electrical condition of the heart.

It would be expedient to introduce some measures designed to reduce myocardial stimulation in the first stages of the operation upon the appearance of large groups or allohythmic type of extrasystolic contractions.

3) Various extrasystoles, group-type and polytopic, occur almost according to a pattern during the manipulations in the area of the pericardium, left appendage, atrium cordis and particularly the ventricle. Large groups of polytopic ventricular extrasystoles appear at the time of the commissure incisions; myocardial nutrition is usually not affected. After this stage, the electrical picture of the heart becomes relatively stable, and frequently the initial picture is gradually restored by the end of the operation. Particular attention should be focused on myocardial nutrition immediately after the mitral commissure incision with a view to detecting a possible development of an acute left ventricular strain. /141

4) Interference with dissociation, migration of the cardiac pacemaker and atrioventricular rhythm are observed fairly frequently. The disruption of intra-ventricular conduction is infrequent. The appearance of large groups of nontypical ventricular oscillations and particularly paroxysms of ventricular tachycardia against a background of disrupted myocardial nutrition (Fig. 45) are unfavorable from a prognostic point of view. In such cases, the prevention of the development of ventricular flutter or fibrillation should be borne in mind.

5) At any time during the surgery, particularly after the commissurotomy incision, a decrease in the frequency of the cardiac rhythm, even if it does not exceed the limits of sinus bradycardia, should alert the physician to the danger of a cardiac arrest.

A commissurotomy is a mechanical operation on the heart that produces enormous changes in the hemodynamics. The surgeon in this case plays the responsible role of a regulator of pathological hemodynamics. A successful operation is to a large extent determined by a justifiable decision to operate and a good preoperative preparation. It is very important to determine the functional condition of the heart before the operation, its contractive energy and the myocardial reserve. But the outcome of a technically successful operation actually depends on the anatomic and functional possibilities of the myocardium.

11. Coronary Insufficiency

Coronary insufficiency is a pathophysiological concept implying a disrupted normal balance between the oxygen supply to the myocardium and its actual needs. It is one of the major causes of ischemia in the myocardium, and the extent of ischemia, as was well illustrated by Williams (1960), depends on a number of factors determining the supply of oxygen to the myocardium (the oxygen content in the arterial blood, coronary blood flow, hypertrophy, hemoglobin concentration, blood pressure, etc.)

Such a disruption of the physiological mechanisms governing the cardiac supply frequently leads to the development of crude pathomorphological changes.

The development of coronary insufficiency may be due to a variety of causes, such morphological factors as coronary sclerosis, thrombosis or embolism of the coronary vessels, coronaritis, hemorrhages into the walls of these vessels, etc. These factors serve to disrupt the quantitative blood supply to the myocardium, that is, they actually reduce the volume of the coronary blood flow. Behaving in similar ways also are such factors as pronounced tachycardia, and a heavy overload of the heart, etc., whereby the absolute quantity of the coronary blood flow, though unchanged, does not meet the increased requirements of the myocardium supply. There are also other factors, such as pronounced anemia whereby the quality of the blood composition is changed but its quantity does not reveal any noticeable change. The neural factor, which is conducive to the development of a coronary vasospasm, plays an important part in all the mentioned mechanisms. /142 /143 /144

A number of clinical classifications of coronary insufficiency have been proposed. We find that the most suitable classification, from a clinico-electrocardiographic point of view, is one that defines the acute and chronic coronary insufficiency which may or may not be accompanied by an infarct (with a stenocardia syndrome) or myocardial infarction.

In the case of coronary insufficiency, particularly when accompanied by

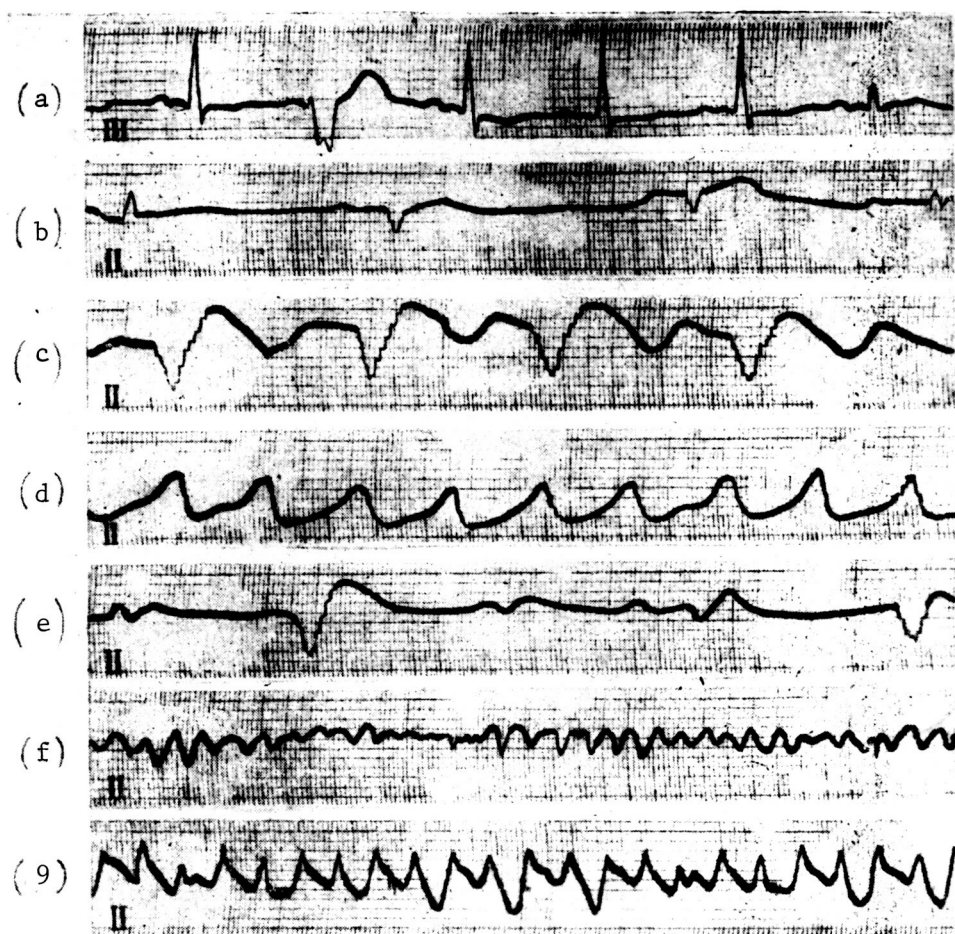


Fig. 45. Dynamic electrocardiographic observation of cardiac operation connected with mitral stenosis (operation performed on a clinically dead female patient). Electrocardiograms of patient M. V., age 36, with following diagnosis: mitral defect with prevalent stenosis. (a) Before anesthesia: deterioration of food intake, frequent extrasystoles, some of them of the fusion-beat type, and aberrant ventricular complexes; (b) a considerable decrease in rhythm frequency and deterioration of atrioventricular conduction; (c) ventricular rhythm; (d) ventricular tachycardia; (e) less frequent rhythm and considerable change of complexes; (f) a digital dilatation of the mitral opening is followed by ventricular fibrillation; (g) the heart is massaged after a repeated defibrillation (see continuation).

myocardial infarction, the physico-chemical processes in the damaged myocardial zone undergo considerable changes, and the result is that the cellular membrane is deprived of its nonconductive characteristics and the extent of its polarization is reduced. Reynolds and Van der Ark, (1959) point out that the ligation of the coronary artery accelerates the restoration process in the deep layers of the damaged area, and this is occasionally observable in the upper layers also. Sodi-Pallares (1956) writes that various de-

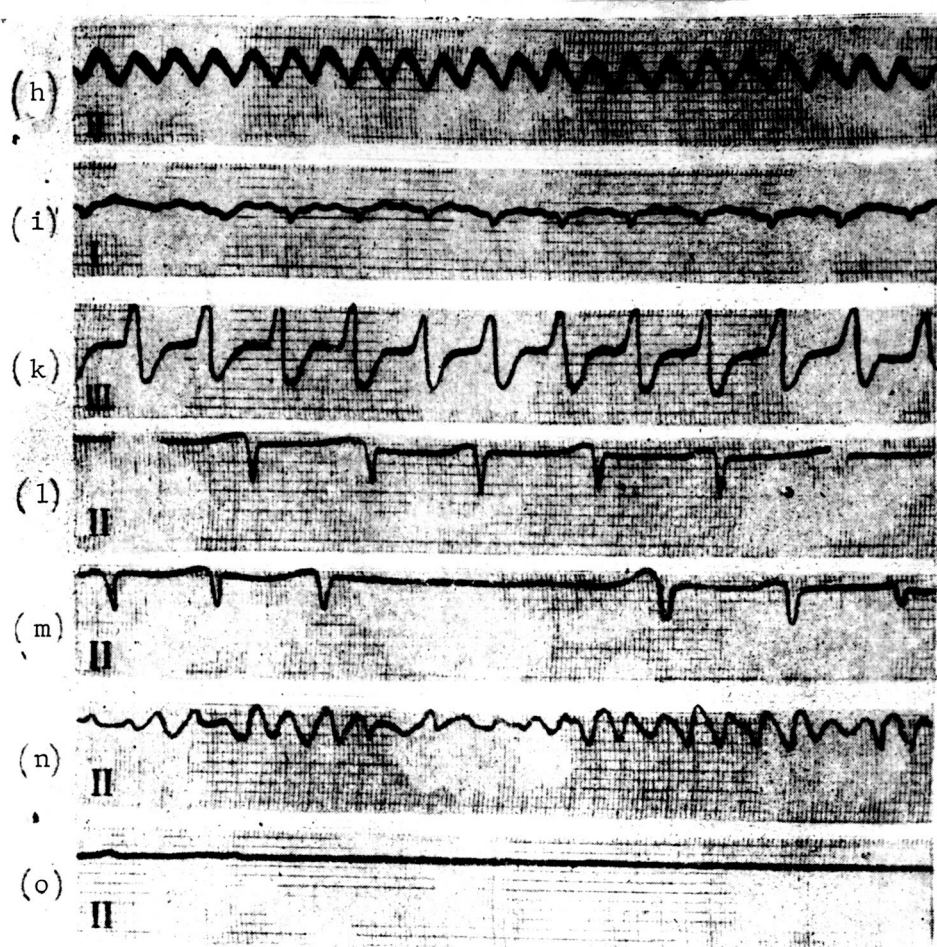


Fig. 45 (continued). (h) ventricular flutter; (i) independent cardiac sinus /143 rhythm follows defibrillation and lengthy massage; (k) ventricular rhythm; (l) reduced frequency of ventricular rhythm; (m) periodic cardiac arrest of short duration; (n) ventricular fibrillation after a lengthy massage; (o) cardiac arrest.

degrees of myocardial damage produce three different electrocardiographic patterns characteristic of slight injury, more serious injury and tissue necrosis. Within the same zone of injury, these degrees correspond to the zones of ischemia, injury and necrosis (these terms are used in an electrocardiological sense). On the basis of his numerous experimental observations of dogs, Bailey (1943, 1958), one of the initiators of this viewpoint believes that the center of the damaged area contains a necrotic zone (dead zone) with a zone of injury arranged concentrically around it and an ischemic zone developing in the periphery (Fig. 46); eventually the zone of injury disappears and the zone of ischemia is found in the periphery of the necrotic zone.

Following is an electrocardiological description of the zones mentioned.

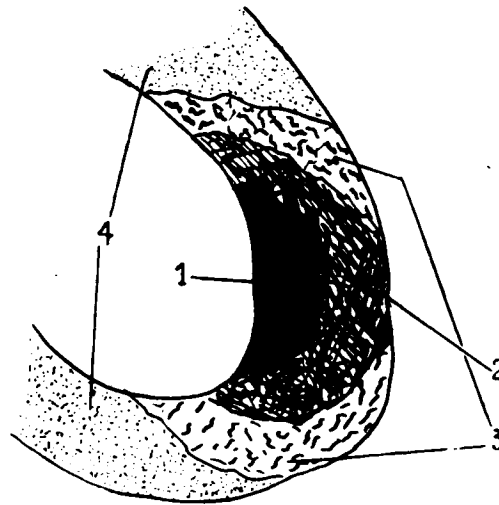


Fig. 46. The three zones of the myocardial injury area, according to Bailey. 1. Zone of necrosis; 2. zone of injury, 3. zone of ischemia; 4. healthy muscle. The necrotic focus is on the endocardial surface of the myocardium.

No histological changes are found in the zone of ischemia, the polarization of the cells during the diastole is normal from an electrophysiological point of view, the depolarization process is as usual, and there is some retardation of the repolarization process. The electrocardiogram records an extended Q-T interval and an inverted T wave. An important feature of these changes is the location of the zone of ischemia in relation to the sub-endocardial or subepicardial layers of the myocardium.

Somewhat blurred and reversible histological changes are observable in the damage zone. The cell polarization of this zone during the diastole is incomplete from an electrophysiological point of view, the depolarization process is somewhat retarded, and the repolarization process is also incomplete. Some of the cells become refractory and are not included in the depolarization process during the expansion of the stimulating wave. The result is the development of a so-called diastolic current of the injury between the healthy and injured portions of the myocardium at rest, and the electrocardiogram records a displacement of the isoelectric line or rather a displacement of the T-P or T-R segment from the isoelectric line. The healthy section is stimulated and becomes negative during the depolarization process, while the zone of injury is not depolarized and reveals a positive charge in relation to the stimulated portion. Thus the systolic current of the injury developing during the stimulation process produces a displacement of the RS-T segment on the electrocardiogram. Important also is the fact that the zone of injury is arranged subendocardially or subepicardially. On the whole, it may be said that the electrocardiographic expression of the injury consists of a single-phase curve where the QRS complex, the RS-T segment and T wave comprise one common characteristic wave. /145

Crude histological changes develop in the zone of necrosis. From an electrophysiological point of view the necrotic zone is a dead zone where the cells are not polarized during the diastole, are not capable of stimulation and no electric phenomena occur in the process of stimulation. The necrotic zone is capable only of passing electrical forces, and, if it is arranged transmurally, the electrode opposite that zone will record the intracavitary potential of the appropriate ventricle (the so-called "electric window" potential). Other phenomena occur (see below) in the case of the subendocardial or subepicardial arrangement of the zone. /146

The above described ischemic theory which explains the electrophysiological manifestations of coronary insufficiency has been fairly thoroughly treated in our literature. Although certain aspects of that theory are subject to criticism and the theory itself is not widely accepted, we have based our study primarily on that theory since it is frequently very useful in practical problems. Attention should be called here to M. G. Udel'nov's interesting theory designed to explain the mechanism governing the development of the single-phase curve. Backed by the results of their original experiments, M. G. Udel'nov and his associates (1958) believe that the formation of a single-phase curve is associated with the chemical processes, particularly the movements of the electrolytes: the potassium content is reduced in the necrotic zone and increased in the surrounding tissue. V. S. Salmanovich's observations (1959) clearly point to the importance of potassium in the mechanism governing the formation of a single phase when a piece of dead muscular tissue is applied to a healthy animal heart.

A. Acute coronary insufficiency without infarction (with a stenocardiac syndrome). Acute myocardial hypoxia or anoxia is electrocardiographically recorded as ischemia and injury, mostly in the subendocardial layers of the antero-lateral wall of the left ventricle. The major changes on the electrocardiogram produced by a severe seizure (Fig. 47) are found in the RS-T segment and T wave; the P wave and the P-Q segment are not subjected to any noticeable or characteristic changes, but the QRS complex is reduced (Kownacki and Kownacki, 1958), the R wave is increased and the S wave reduced in amplitude (Ekmekci, et al., 1961). Discordant and, less often, concordant displacements of the RS-T segment which assumes a pathological (and frequently a plateau-like) form of junction, are observable in the standard leads. Ekmekci et al. (1961) believe that the upward displacement of the RS-T segment is connected with a more pronounced ischemia than its downward displacement. The T wave is reduced and frequently becomes isoelectric, two-phased or negative. Similar changes appear even clearer in the precordial leads. Evans and Pillary (1957) attach some importance to the displacement of the T-U segment, Palmer (1948) finds that the U waves become negative, and the T waves high and symmetrical. Papp (1957) does not attach much importance to the changing U wave in a coronary disease. Extrasystoles, various disruptions of conduction, etc., may develop in the course of an acute seizure.

The above-described changes may not be found in the periods between the seizures, and the various functional tests which are frequently very helpful in diagnosis therefore acquire increasing importance. The tests made /147

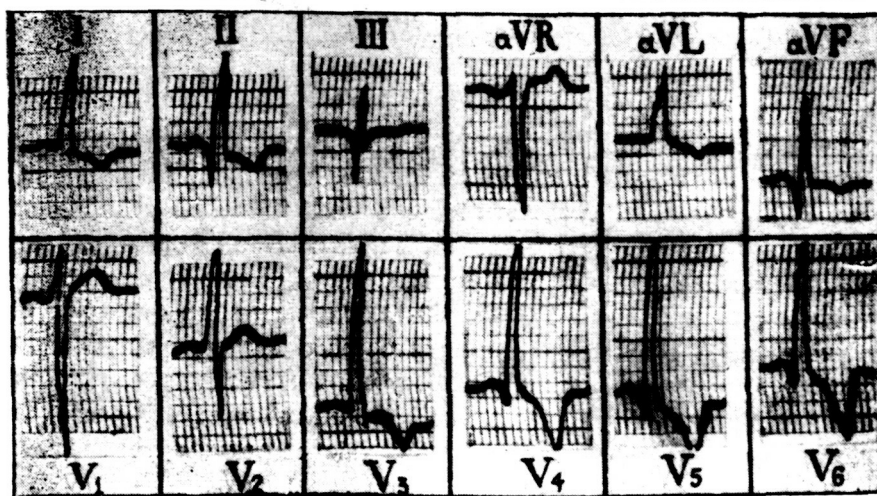


Fig. 47. Acute coronary insufficiency with a stenocardiac syndrome. Electrocardiogram of patient S. A., age 57, with following diagnosis: hypertension, stage IIIa, and atherosclerotic cardiosclerosis. Patient had a stenocardiac seizure during the EKG; there is a considerable disruption in the supply of the myocardium in addition to the hypertrophy and strain of the left ventricle.

for this purpose involve the use of such pharmacological agents as adrenalin, pitressin, nitroglycerin, etc. But medicinal tests are not always practical. We find that a test involving a physical strain, as proposed by Master as far back as 1935, has great advantages. This is more of a physiological test and, as Manning's observations indicate (1957), the correct interpretation of the resulting data may prove very important for diagnostic purposes. Based on this principle is the Nylin cardio respiratory test and its modification involving the use of a three-step portable ladder (Maurea, Nylin and Sollberger, 1958). A number of authors (Levy, Barach and Bruenn, 1938; Coulshed, 1960, etc.) attach considerable importance to the hypoxemia test.

B. Acute coronary insufficiency with myocardial infarction. It should be borne in mind that the following data are very important for the compilation of an appropriate electrocardiographic picture of myocardial infarction: the size of the necrotic focus, its localization, its extent, its relation with the epicardial and endocardial heart surfaces (transmural, subendocardial and intramural), the remoteness of the event as well as the general condition of the heart prior to the myocardial infarction. The dynamics of the electrocardiographic changes produced by a myocardial infarction may be divided into three stages whereby the necrosis and damages are observable in the first stage, and ischemia follows them a little later.

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The first stage usually begins 12-36 hours after a severe attack, and is characterized by the appearance of a monophasic curve (Fig. 48a), where the descending bend of the R wave, after passing a few millimeters, fuses

with the T wave forming a cupola-shaped joint. It should be pointed out, however, that this state is preceded by still another stage which has not been thoroughly studied in the literature. This earliest phase of the development of a myocardial infarction is not observable in practice, or very seldom recorded, as an electrocardiographic examination is usually made several hours after a severe attack. In the case of one patient, we succeeded in observing the following (Fig. 49): an EKG taken two hours after a severe attack recorded high, acuminate and symmetrical T waves and an upward shift of the RS-T segment while the monophasic curve was observed only 18 hours later. This prompts the assumption that the earliest change in the case of a myocardial infarction is the appearance of ischemic T waves. In this connection it is possible to agree with G. Ya. Dekhtyar' (1955) that the development of the necrotic focus in the myocardium should be divided into four rather than three states. He characterizes the first stage as ischemic. V. Ye. Nezlin (1955) also speaks of these ischemic changes, and such changes are indicated by the experimental observations of Lengyel, et al. (1957).

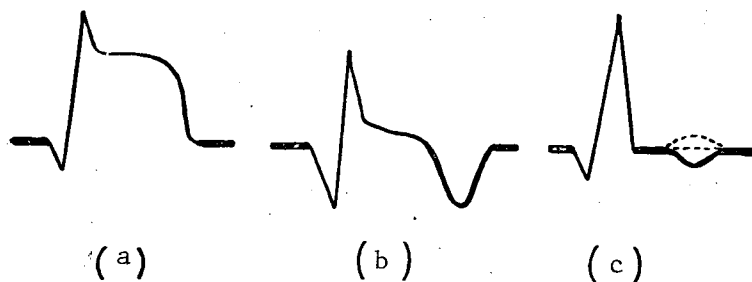


Fig. 48. EKG of various stages of myocardial infarction. (a) First stage; (b) second stage; (c) third state (explanation found in text).

A pathological Q wave is occasionally revealed in the first stage. This stage lasts an average of 12-24 hours, occasionally several days, and is then followed by the second stage.

In the second stage (Fig. 48b), the descending bend of the reduced R-wave gradually goes down to the iso-electric line. At the same time, the dome-shaped RS-T segment is gradually shifted downward, and the T wave becomes negative and symmetrical with an acuminate vertex. The pathological Q wave is always revealed in this stage. The second stage lasts an average of from one to six months.

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The third stage is characterized by the restoration of the initial EKG pictures due to the further cicatrization of the focus and the improvement of the myocardial blood supply. The pathological Q wave usually remains, and the changes of the T wave are frequently restored (Fig. 48c). Gardberg (1957) noted the disappearance of the T wave changes in almost all of the patients by the end of the first year after a severe attack. Examining myocardial infarction patients one year after the onset of the disease, Grittler, Schack and Vesell (1956) found a normal electrocardiogram in only one of them.

Pappas (1958) finds that the pathological Q wave may disappear only in cases when the acute stage of the infarction is relatively light and the arterial pressure is not increased.

Such is the general dynamics of the schematic development of myocardial infarction. Of course, there may be other phenomena. T. B. Korenevskaya (1960), for example, found the S wave in the left precordial leads during an acute state of infarction. The wave disappeared with the general improvement of the patient's condition. A number of authors point to the importance of the U wave, but we agree with Papp (1957) that this U wave cannot be of great diagnostic importance. We follow the above-cited scheme and, along with the other authors, find that if the described dynamics is observable in I, aVL, and precordial leads, then the focus of the infarction is localized on the anterior wall of the left ventricle. If the same dynamics is observable in III (very often also in II) and aVF leads, then the focus is to be found on the posterior wall of the left ventricle. The changes in the other leads, in this case, are discordant in relation to the infarction dynamics in the mentioned leads. Such general treatment of the problem is designed in order to understand the EKG picture in localization of the infarction in the left ventricle.

It should be pointed out that the affection of the subepicardial layers of the myocardium in the case of pericarditis results in the development of infarct-like changes of the ventricular complex. In this case, however, the changes of the RS-T segment and T wave are concordant in all the leads, the Q wave does not appear, and the disappearance of the monophasic curve and appearance of negative T waves are considerably slower.

We agree with Laham (1954) that infarction is schematically divisible into five major types: anterior, inferior or diaphragmatic, lateral, apical and septal. But from a practical scientific point of view, attention should be focused on all the frequent or rare cases of infarct localization occurring in clinical practice. From the proposed classifications of myocardial infarct we shall select the following classification developed by Lenegre, Carouso and Chevalier (1954) (these authors base their classification on Wilson's data). /150

1. Anterior infarction
 - Acute anterior infarction.
 - Anterolateral infarction.
 - Anteroseptal infarction.
 - Apical infarction. /152
2. Posterior infarction.
 - Posterodiaphragmatic infarction.
 - Posterolateral infarction.
 - Posterobasal infarction.
3. Deep septal infarction.
4. High lateral infarction.

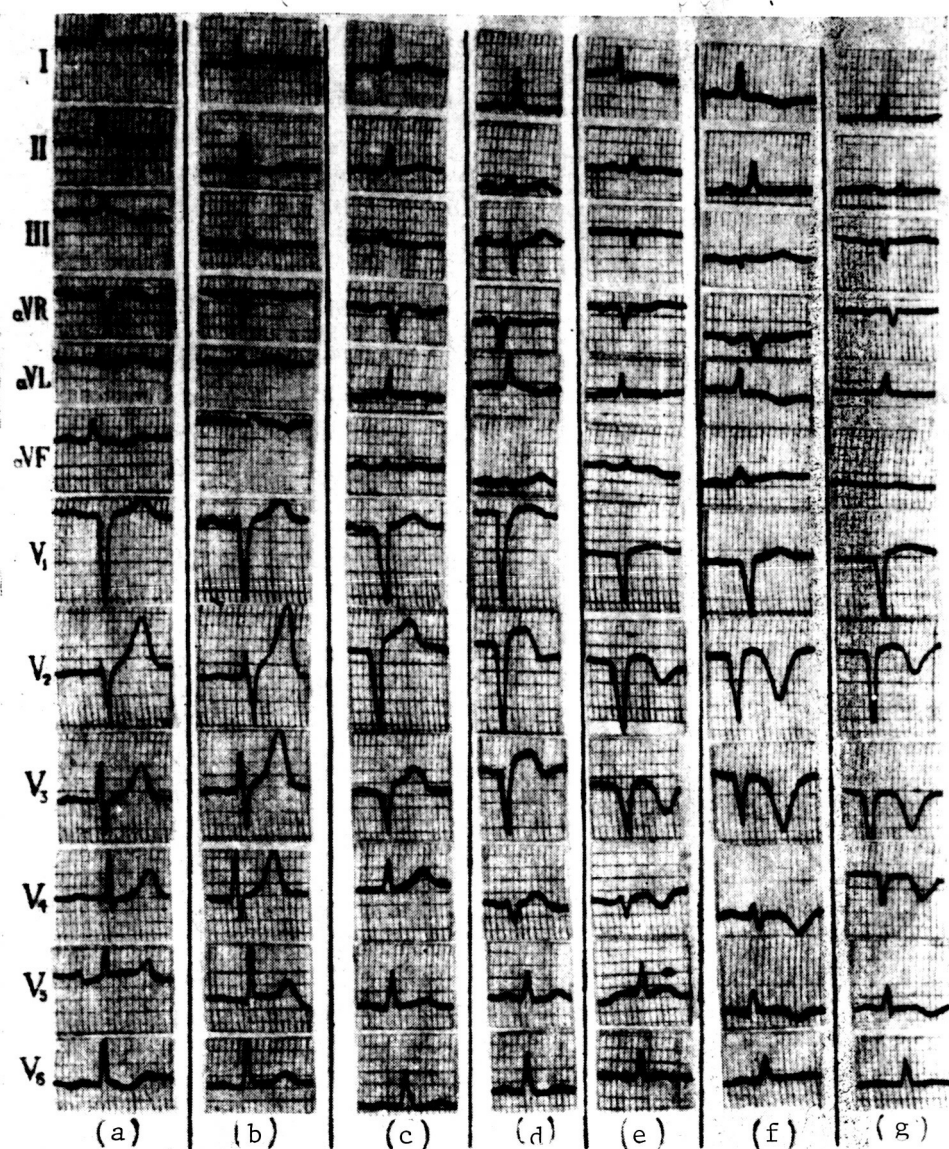


Fig. 49. Anterior infarction. Electrocardiogram of patient S. V., age 48, /151 with following diagnosis: myocardial infarction, atherosclerotic cardiosclerosis and hypertension, Ib degree. Dynamics of electrocardiographic data: (a) ischemia on the posterior wall developing early in the acute seizure, and the T wave in the V_2 lead is symmetrical and increased; (b) high symmetrical waves appear in the V_{2-4} leads two hours later, and the RS-T segment in lead I is shifted upward; (c) a monophasic curve takes shape in the V_{1-3} leads 18 hours later; (d) a typical monophasic curve in the $V_{2,3}$ leads 44 hours later, and the T wave in the aVL lead is negative (see continuation).

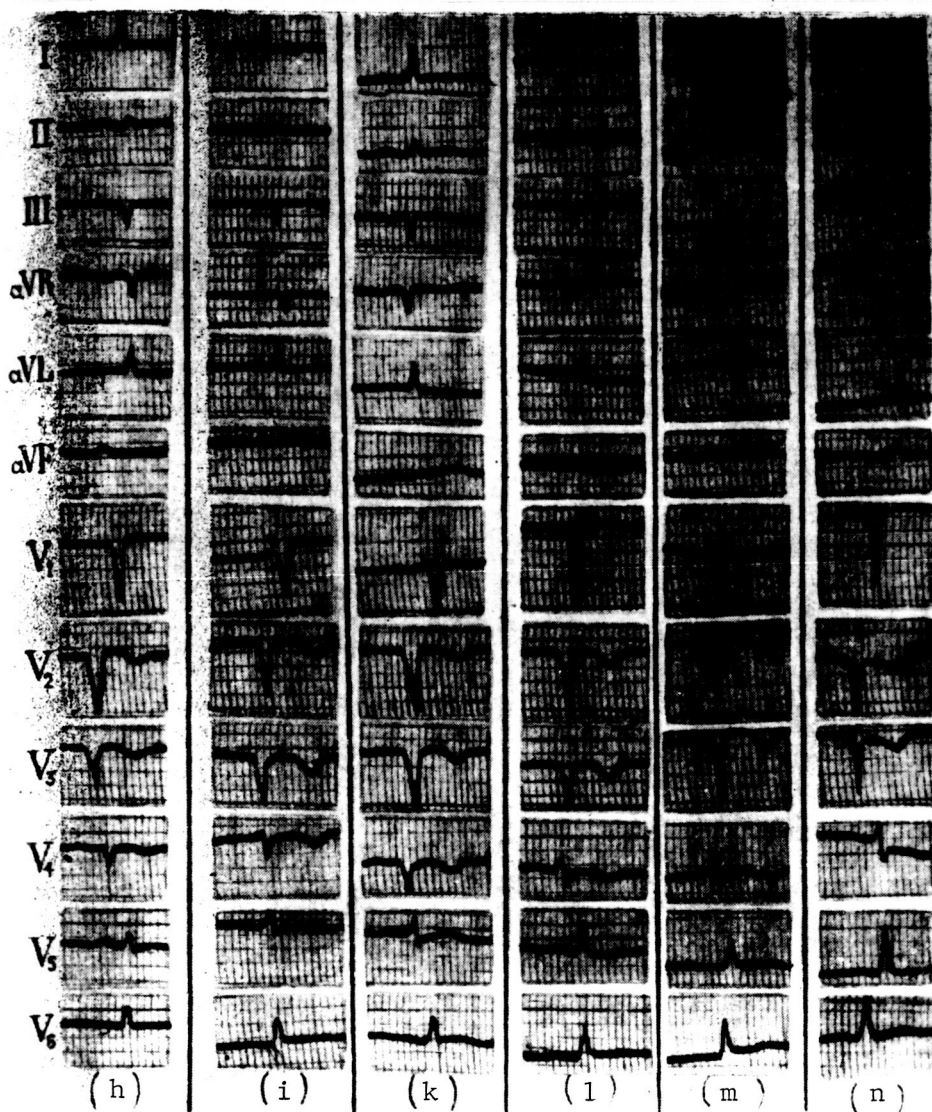


Fig. 49 (continued). Further pictures were taken periodically over a period of two months; the dynamics of an anteroseptal infarction is observable in the I, aVL and V₂₋₄ leads with ischemic events in the lateral wall of the left ventricle.

5. Subendocardial infarction.
6. Multiple infarctions.
7. Right ventricular infarction.
8. Auricular infarction.

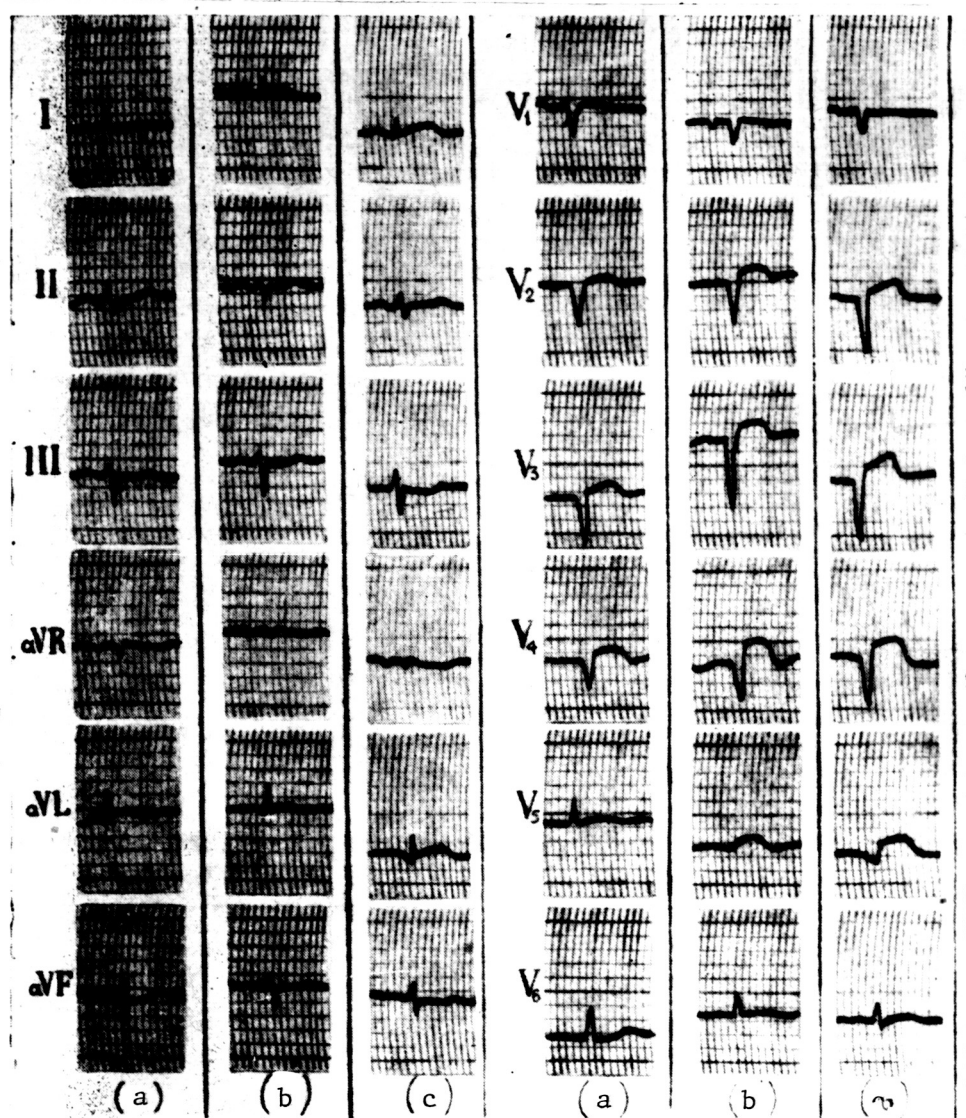


Fig. 50. Anterior infarction. Electrocardiograms of patient E. G., age 48, /153 with following diagnosis: myocardial infarction, atherosclerotic cardio-sclerosis. (a) First day after a severe seizure: antero-septal infarction; (b) a day later: infarction pattern observable in all precordial leads, a cupola-shaped upward shift of the RS-T segment in lead I, the infarction changes become more descriptive in the aVL lead; (c) five days later: typical picture of a diffuse anterior infarction. It is interesting to point out that a picture of auricular infarction was observed three days later: the P-Q segment shifted downward in the I and II lead, and upward in the V₁ lead. The patient died a month later of a repeated infarction which manifested itself in the form of a paroxysmal tachycardia.

9. Infarction combined with disrupted intraventricular conduction.
Anterior or posterior infarction combined with a right bundle branch block.
Myocardial infarction combined with a left bundle branch block.

Infarction combined with a local intraventricular block.

10. Infarction accompanied by a disrupted cardiac rhythm.
11. Cardiac aneurysm.

Following is a brief description of an EKG taken of such forms of myocardial infarction.

a. Anterior infarction. In an acute anterior infarction (Fig. 50), the dynamics of the infarction is observable in the precordial leads from V_1 to V_6

and in the aVL and I leads. In the initial state of the infarction, the RS-T segment is shifted upwards in a dome-shaped manner in lead I, and downward in lead III. The second stage is characterized by the appearance of a negative symmetrical T wave in lead I, and a positive wave in lead III. The pathological Q wave appears in lead I as soon as it is noted in lead aVL. The aVR lead presents the usual picture, but the T wave is frequently positive; a pronounced R wave occasionally comes into view (V. Ye. Nezlin and S. Ye. Karpay, 1959). The aVL lead reveals an upward shift of the RS-T segment followed by the appearance of a typical pathological Q wave (this is observable also in the early stage of the infarction) and a negative symmetrical T wave. There may be a slight downward displacement of the RS-T segment in the aVF lead, and the T wave is usually positive. In the first stage the precordial V_{1-6} leads reveal a typical upward-displaced dome-shaped RS-T

segment with QS or QR type complexes. Later, when the RS-T segment is lowered, deep symmetrical negative T waves make their appearance.

In an anterolateral infarction the EKG records almost the same picture /154 as in the previous form, but in the precordial leads the displacement of the RS-T segment, QS or QR type complexes and coronary T-waves appear primarily in the $V_{5,6}$ positions.

In an anteroapical infarction (Fig. 49), the standard or amplified unipolar limb leads do not usually reveal any characteristic symptoms of an infarction. An initial dome-shaped upward displacement of the RS-T segment and, a little later, coronary negative T waves appear in the precordial V_{1-4} leads. Observable in these same leads are QR or QS complexes

and, as Richman, Wolff and Call (1957) point out, the QS form is very typical of an infarction localized in the area of the septum adjoining the anterior wall. It should be recalled, that these phenomena are not observed in the V_{1-6} leads.

An apical infarction reveals a characteristic drop in the QRS voltage and

a frequently concordant upward shift of the RS-T segment in all the three standard leads. The pathological Q wave is not recorded in lead I, nor are characteristic infarction symptoms observable in the reinforced unipolar leads from the extremities. A high R wave amplitude is occasionally found in the aVR lead while the aVF lead shows an upward shift of the RS-T segment which is later followed by the formation of a negative T wave. The precordial $V_{4,5}$ leads reveal the appearance of QR or QS type complexes, an upward shift of the RS-T segment and the eventual formation of negative T waves.

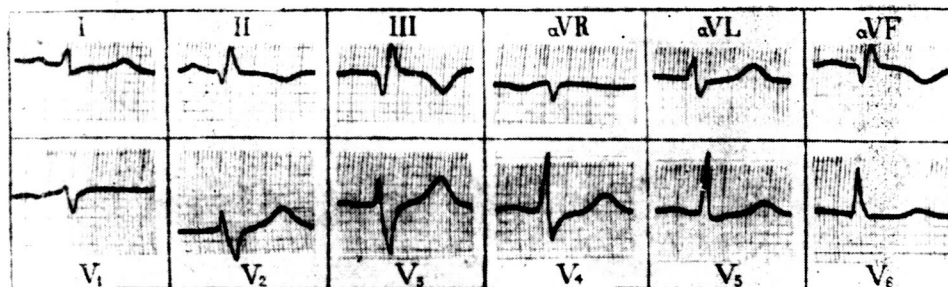


Fig. 51. Posterior infarction. Electrocardiogram of patient T. V., age 50, with following diagnosis: myocardial infarction, atherosclerotic cardiovascular disease. Posterodiaphragmatic infarction, first stage changing to second. Infarction produced changes are pronounced in the II, III and aVF leads; discordant phenomena are noted in the I, aVL and V_{4-6} leads.

b. Posterior infarction. The first stage of a posterodiaphragmatic infarction (Fig. 51) is characterized by the appearance of a typical monophasic curve in leads III and II where it is relatively less pronounced. A discordant lowering of the RS-T segment is observable in lead I. Eventually, the RS-T segment is gradually lowered to the isoelectric line in leads III and II, and deep negative T waves appear, while lead I reveals an increasing amplitude of the positive T wave. The pathological Q wave is clearly visible in lead III and with a relatively lower amplitude also in lead II. The changes typical of infarction appear in the aVF of the amplified unipolar limb leads. The ventricular complexes in this lead acquire a QR or QS form, the RS-T segment is at first raised but later lowered to the isoelectric line, and negative T waves appear. Direct indications of an infarction are not found in the precordial leads. A downward shift of the RS-T segment which later becomes isoelectric is frequently observable in the right and transitional leads. In that period the T waves grow tall and symmetrical. Occasionally the R wave in these leads increases its amplitude in the initial stage of the infarction. Wachtel and Teich (1956) find that the appearance of high acuminated T waves in the V_{2-5} leads is the earliest symptom of

diaphragmatic infarction.

The picture of a posterolateral infarction is similar to that of a posterodiaphragmatic infarction, but changes typical of an infarction are observable

not only in the III, II and aVF leads, but also in the precordial $V_{5,6}$ leads.

In a posterobasal infarction, the standard leads as well as the amplified unipolar leads from the extremities are not subjected to any changes, and the precordial V_{1-3} leads reveal only indirect symptoms, such as the lower-

ing of the RS-T segment from the isoelectric line and the rising amplitude of the T wave which acquires a symmetrical form with a sharp peak. Definite diagnostic importance attaches to the esophageal leads. But it is very difficult to diagnose such a localization of the infarction, and an accurate diagnosis can be made only on the basis of a good clinical and electrocardiographic analysis.

c. A deep septal infarction. Symptoms of an anterior as well as a posterodiaphragmatic infarction appear in this form of the disease, as the necrotic focus is located along the entire interventricular septum, from the anterior to the posterior cardiac surface. Thus in the initial stage of the process there is a typical elevation of the RS-T segment which eventually descends gradually and is followed by the appearance of deep symmetrical negative T waves in the III, II, and aVF limb leads and in the V_{1-4}

precordial leads, even when the infarction extends to the lateral sections of the left ventricle. Pathological Q waves are found in all the above-listed leads. QS or QR-type ventricular complexes are observable in the precordial leads. Abrahams (1957) describes the case of septal infarction in which the /156 changes affected only the T waves and disappeared when the patient's condition improved.

d. A high lateral infarction. The electrocardiographic changes characterizing such an infarction are observable only in the aVL lead. In some cases uncharacteristic changes of the T wave may be found in lead I. As the location of the necrotic focus is high, the ordinary precordial leads do not detect it, and only special precordial leads with an investigating electrode in the second or third intercostal area along the axillary lines can detect it.

e. Subendocardial infarction. A stenocardiac seizure may occasionally produce a subendocardial infarct with a developing myocardial damage in the deep subendocardial layers. If the injury is in the area of the anterolateral wall of the left ventricle, the precordial V_{3-6} leads reveal a dis-

placement of the RS-T segment down from the isoelectric line and negative T waves. If the damage is localized in the area of the posterior wall of the left ventricle, which is observable less often, the downward shift of the RS-T segment and the negative T waves are recorded in the III, II and aVF leads. It is characteristic in this connection that pathological Q waves are not detected in this form of infarction. Durrer et al. (1961) believe that the occasionally observable T wave is a result of a reduced potential produced by the stimulation of these sections. Thus a diagnosis of a subendocardial infarction may be made only in the case of clinically manifested stenocardiac seizure and if the above-described electrocardiographic changes

are detected also in repeated examinations. A differential diagnosis should take account of the fact that such an electrocardiographic picture may be observable also in pulmonary embolism, shock and digitalis therapy. An electrocardiogram of a patient suffering from subendocardial infarct is cited in Fig. 52. What makes this case interesting is that the picture was observed after a severe seizure of paroxysmal tachycardia of a neurogenic origin.

Unlike the subendocardial infarction, subepicardial infarction reveals a pathological Q wave, a decreasing R wave, an upward shift of the RS-T segment and a negative T wave. A minor intramural infarct produces only a few changes in the RS-T segment or T wave as a result of the ischemia or damage of the neighboring sections. Other cases may reveal pathological Q waves and negative T waves. A transmural infarction is characterized by a typical infarct curve.

f. Multiple infarctions. The development of such infarctions may be simultaneous or consecutive. An electrocardiographic diagnosis of the frequently observable consecutive development of multiple infarction is very difficult as the fresh infarction covers the picture of the old infarction or, conversely, it cannot be seen against the background of the old infarction. Anamnestic and clinical data and repeated electrocardiographic examinations are helpful in such cases. The simultaneous development of multiple infarctions is characterized primarily by infarctions of the anterior and posterior walls of the left ventricle (Fig. 53). This case reveals an electrocardiographic picture of a deep septal infarction, but characteristic infarction changes are observable also in the precordial V₄₋₆ leads. M. B. Tartakovskiy /158

(1958) writes that on rare occasions the anteroposterior infarctions are not recorded on the EKG because of the disappearing "bioelectrical asymmetry" which is required for diagnosing an infarction.

g. Infarction of the right ventricle. The fact that an isolated infarction of the right ventricle is observable on very rare occasions is, according to Lenegre, Carouso and Chevalier (1954), due to its thin wall as well as the very low intracavitary pressure (25-30 mm Hg with 120-125 mm in the left ventricle). The extension of the infarction of the left ventricle to the neighboring sections of the right ventricle is frequently observed in actual practice. It is practically impossible to make an electrocardiographic diagnosis of an infarction of the right ventricle because the ventricular complexes in the leads from the epicardial surface and in the right ventricular cavity are indistinguishable from one another. /159

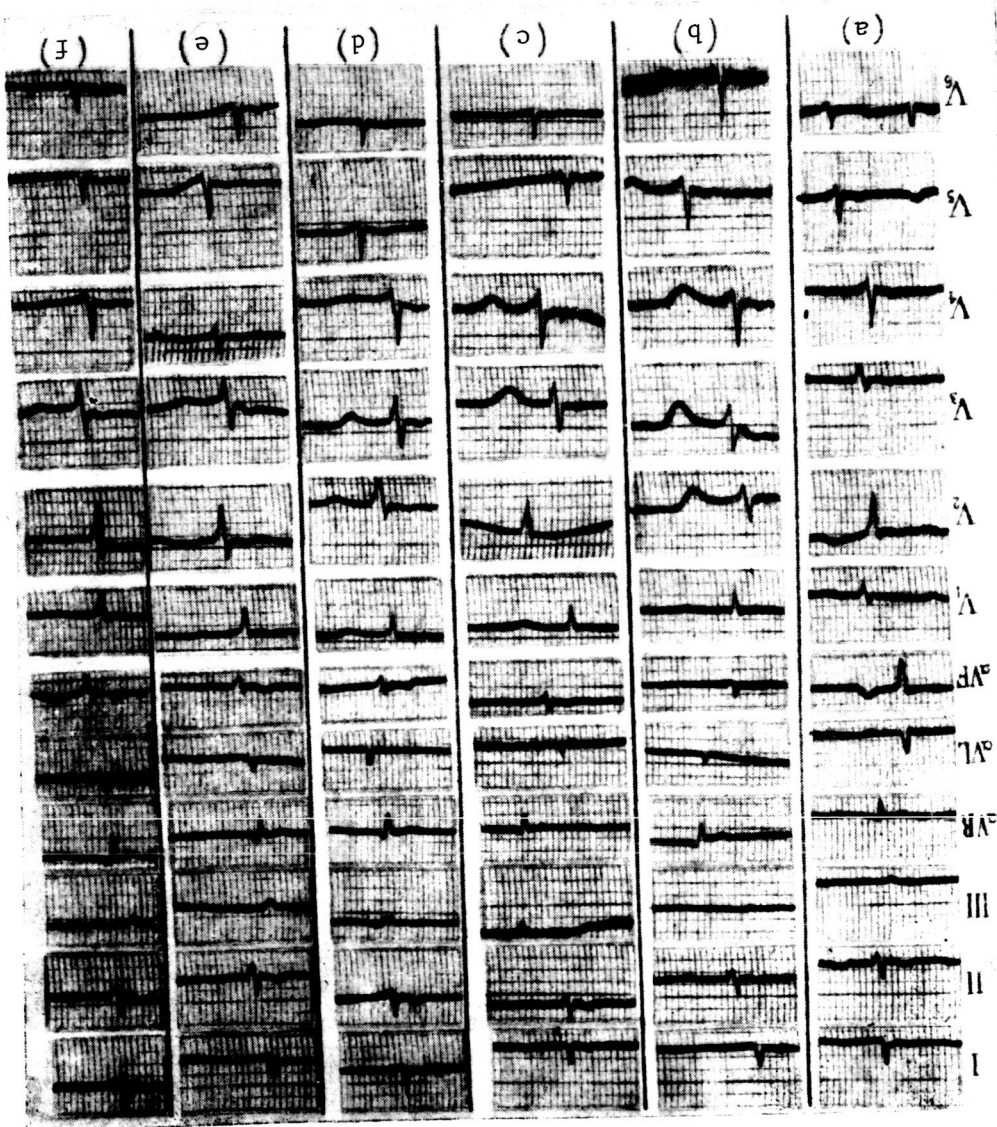
h. Auricular infarction. An auricular infarction is more frequently found to be combined with a ventricular infarction than isolated. An electrocardiographic diagnosis of an auricular infarction is difficult as such cases do not reveal the type of direct and typical changes observable in ventricular infarct. In the case of a clinical picture of a myocardial infarction, however, certain electrocardiographic changes acquire diagnostic importance. From this point of view, we must not lose sight of the various disruptions of the auricular rhythm accompanying the clinical manifestation of a seizure, such as auricular extrasystoles, auricular tachycardia, fibrillation or

flutter, the migration of the cardiac rhythm conductor, etc. Of definite importance also is the displacement of the P-Q segment (Fig. 54). This segment is shifted upwards in lead I and downward in lead III in an infarction of the

in repeated investigations. The Q wave is not detected.

RS-T segment and negative T waves are observable in the V₂₋₅ leads and found are: (c) 11/4/62, (d) 11/6/62, (e) 11/13/62 and (f) 11/17/62. A displaced the day after the attack of paroxysmal tachycardia; the dates of the EKG's topic extrasystoles are outlined on the film; (b) EKG recorded on 11/2/62, ed on 10/31/62 - before the attack of paroxysmal tachycardia (frequent poly-nounced cardiovascular syndrome and paroxysmal tachycardia: (a) curve recorded-36, with following diagnosis: myocardial dystrophy, neurasthenia with pro-

Fig. 52. Subendocardial infarction. Electrocardiogram of patient S. L., age/157



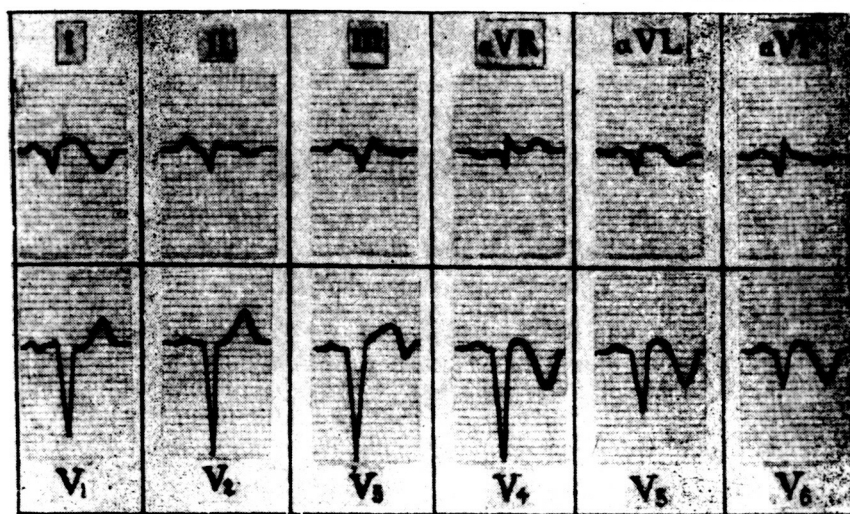


Fig. 53. Multiple infarctions. Electrocardiogram of patient S. G., age 58, with the following diagnosis: Atherosclerotic cardiosclerosis and myocardial infarction. Symptoms of anterior and posterior myocardial infarctions in the second stage are found on the EKG.

anterior auricular wall, while an infarction of the posterior wall reveals a reverse picture. The upward shift of the P-Ta segment by more than 0.5 mm in the V_5 and V_6 leads and its corresponding descent in the V_1 and V_2 leads

are, according to Liu, Greenspan and Piccirillo (1961), the major symptoms of an auricular infarction, as are also the upward shift of the P-Ta segment by more than 0.5 mm in lead I and its descent in the II and III leads, and the downward shift of the P-Ta segment by more than 1.5 mm in the precordial leads and by 1.2 mm in the three standard leads accompanied by some form of auricular arrhythmia. They find that the upward shift of the P-Ta segment without its corresponding descent in other leads cannot be interpreted as a symptom of auricular infarction. These authors point to the appearance of changed P waves as an auxiliary symptom of auricular infarction.

i. A combination of infarction and disrupted intraventricular conduction. Myocardial infarction may be combined with a bundle branch block in which case such a block may precede the infarction, develop simultaneously with the infarction or follow it. The first version, whereby the block precedes the development of the infarct, is frequently observable in actual practice.

An infarction of the anterior wall combined with a right bundle branch block produces characteristic changes in the precordial leads. In the case of an anteroseptal infarction, the rSR' type complexes in the V_{1-4} leads, character-

istic of a right bundle block, are replaced by QR type complexes and, unlike the Q wave occasionally observable also without a myocardial infarction in the case of a right branch block in the $V_{1,2}$ leads, the Q wave here is

widened by more than 0.03 sec. The RS-T segment is somewhat displaced, and the dynamics of this displacement are particularly prominent in the repeated examinations. If the infarction extends to the lateral sections of the left /160 ventricle, the Q wave appears also in the $V_{5,6}$ leads, and the changes characterizing the infarction are detected also in the I and aVL leads.

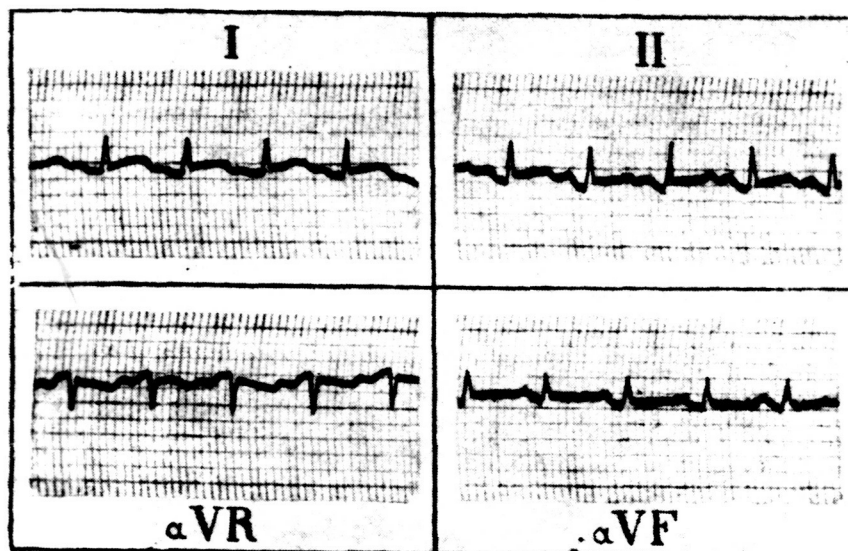


Fig. 54. Auricular infarction. Electrocardiogram of patient B. L., age 22, with the following diagnosis: Mitral defect with prevalent stenosis. Noted on the second day after the mitral commissure incision is a downward shift of the P-Q segment in the I, II and aVF leads, and an upward shift in the aVR lead.

In the case of an infarction of the posterior wall combined with a right bundle branch block, the changes characteristic of an infarction are observable in the III, II and aVF leads. The picture in the precordial leads is characteristic of a right bundle branch block.

It is very difficult to diagnose a myocardial infarction combined with a left bundle branch block. The point is that with such a block, the intracavitary potential of the left ventricle is recorded in the form of an RS complex, and the Q wave characteristic of the infarction cannot be detected. Besides, in the case of a left branch block, the RS-type complexes are observable in the V_{1-4} leads even without an infarction. But the detection of cer-

tain symptoms may prompt the assumption of such a combination. Lenegre, Carouso and Chevalier (1954) believe that because of the systolic current of the injury the RS-T segment assumes an inverse convexity and shifts in a reverse direction in relation to the direction observable in the case of an isolated block of the left ventricle. Moreover, the presence of the Q wave in the left

precordial leads may be a fairly reliable symptom of septal infarct, and the /161
acumination of the Q or QS wave in the $V_{3,4}$ leads is possible in this case.

The described symptoms help to diagnose a myocardial infarction combined with a left bundle branch block, although Besoain, Santander and Gomez-Ebensperguer (1960) have their doubts about these symptoms.

In the case of an infarction with a local intraventricular block (peri-infarction block according to American authors), the subendocardial layers reveal the presence of a large necrotic focus which gradually narrows in the direction of the epicardial heart surface. The result of such a picture is the disruption of the normal extension of the stimulative process in the ventricle, and there is some delay in the stimulation of the individual sections of the epicardial surface of the myocardium. Wide and acuminate Q waves are found in the precordial leads, the R wave is reduced and pointed, and the time of the origin of the internal deviation is increased to 0.07 - 0.08 sec. Such a picture may be produced also by a bundle branch block, but the characteristic feature of the local intraventricular block is the considerable difference in the width of the R wave and particularly the time of the origin of the internal deviation in the neighboring precordial positions. The highest indicators are observable in the V_{3-5} leads.

An infarction with a disrupted cardiac rhythm. A myocardial infarction may lead to various disruptions of the functions of automatism, myocardial conduction and stimulation. Such disruptions include various types of tachycardia and extrasystoles, auricular fibrillation or flutter, various blocks on all levels of the conductive system, etc. According to B. I. Gorokhovskiy (1958), fibrillating arrhythmia is the most common type of arrhythmia observable in myocardial infarction. An infarction is occasionally preceded by a disruption of the rhythm. Katz, Berk and Mayman (1958), for example, describe two cases in which occasional ventricular extrasystoles were detected before the appearance of a typical electrocardiographic picture of an infarction. In rare cases, a myocardial infarction does not manifest itself as a characteristic electrocardiographic picture but as a disruption of the mentioned cardiac functions. Thus, in one case a definite clinical picture of an infarction was recorded on the EKG as only an auricular fibrillation, and in another case the EKG revealed a picture of a left bundle branch block.

The described cases of arrhythmia may occur regardless of the localization of the infarct, but they frequently develop when the infarction affects the posterior wall of the left ventricle, as in this case the process frequently involves also the upper section of the intraventricular septum and disrupts the blood circulation in the ramus septi fibrosi which supplies the cardiac conduction system. It should be pointed out that the development /162
of the mentioned types of arrhythmia aggravates the course of the disease.

1. Cardiac aneurysm. Occasionally following a myocardial infarction, this condition can also be detected on an electrocardiogram. The most char-

acteristic symptom of this condition is the fact that the pronounced Q wave observable in the late infarction stages is accompanied by an upward shift of the RS-T segment, frequently dome-shaped, which is peculiar to the first infarction stage. This displacement is noted in the leads reflecting the localization of the infarction. Thus, in addition to the pathological Q wave in leads I and aVL and the ventricular complexes of the QS type in the precordial leads, in the case of aneurysm of the anterior wall of the left ventricle, we also find (in the same leads) a dome-shaped upward shift of the RS-T segment (Fig. 55). In the case of the aneurysm of the posterior wall of the left ventricle, the pathological Q wave and the dome-shaped elevation of the RS-T segment are observable in the III, aVF and II leads.

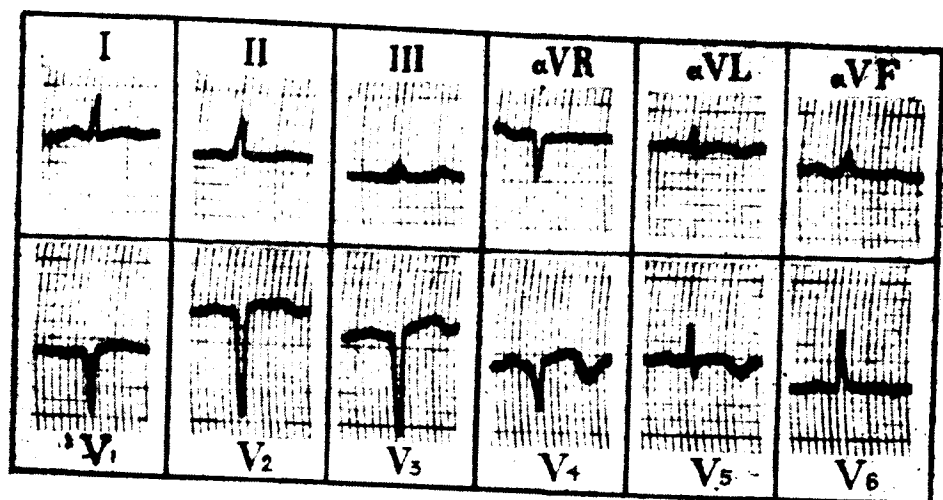


Fig. 55. Cardiac aneurysm. Electrocardiogram of patient M. S., age 42, with following diagnosis: antero-septal myocardial infarction and hypertension, stage IIa. The curve was recorded 6 months after a severe attack: the QS-type complexes and dome-shaped upward shift of the RS-T segment in the V_{1-4} leads, two-phased T waves in the $V_{2,3}$ leads and negative T waves in the I, aVL and $V_{4,5}$ leads.

C. Chronic coronary insufficiency. This is a clinical concept, and therefore can only be defined by a parallel clinico-electrocardiographic analysis. In this case, the electrocardiographic data characteristic of the main disease are accompanied by changes in the ventricular complex, particularly the RS-T segment and T wave as well as various disruptions of the cardiac function. The prominence of these symptoms and the clinical manifestations suggest the existence of a number of clinico-electrocardiographic degrees of chronic coronary insufficiency. We believe it is wrong to base the classification on the electrocardiographic data alone. In our own practice, we do not indicate the degree of chronic coronary insufficiency, and do not even write the word "chronic" in our EKG reports. Z. Z. Dorofeyeva (1956) offers a better classification in this connection. She does not describe the electrocardiographic stages of chronic coronary in-

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sufficiency but does discuss the changes in the electric cardiac activity appearing in the various stages of the development of coronary atherosclerosis, according to A. L. Myasnikov (1956). Thus in the first (ischemic) stage we see primarily a displacement of the RS-T segment and change of the T wave; in the second (thrombonecrotic) stage we find a picture of focal changes with typical or untypical changes in the QRS complex, RS-T segment and T wave and various disruptions of the cardiac functions; in the third (fibrous) stage we observe the stable changes of an advanced cardiosclerosis: cicatricial changes, fibrillating arrhythmia, blocks, etc.

The following functional tests play an important part in the detection of the earliest stages of the development of latent forms of coronary insufficiency: Masters two-step test, the hypoxia test, the sugar test, etc. Our observations have shown that the earliest symptom of coronary insufficiency is the appearance of high, symmetrical and acuminate T waves, especially in the precordial leads. We believe such a manifestation of myocardial hypoxia to be the initial period of coronary insufficiency. To R. P. Stamboltsyan (1957) the presence of such T waves along with a saddle-like upward displacement of the RS-T segment is indicative of coronary angioneurosis, and Freundlich (1956) finds similar T waves in patients suffering from frequent stenocardiac seizures but revealing no other changes on the EKG.

X. CONCLUSION

It is already evident from the contents of this chapter that electrocardiography is the leading method in the field of electrocardiology and is also the starting point for all present-day cardiological knowledge. Any pathologic process in the myocardium alters the normal course of the excitation process in the myocardium and hence necessarily is reflected on the electrocardiogram. The value of this method is especially great in the study of the automatic functions, excitability, and conductivity, when practically all-inclusive responsibility is given this method. In studying hypertrophy and coronary deficiencies this method is of invaluable assistance to the clinician and to the researcher. Electrocardiography is of great value in experimental studies and in particular in cardiac surgery where very often it is the primary indicator for operations under complex conditions. /164

We believe that the pathologic changes in electrocardiograms have been studied sufficiently well, but the several variants of the normal picture, in particular from the point of view of the aging characteristics, have not been sufficiently studied up to the present time. In the subsequent development of this method, work should be done on the experimental study of the characteristics of the propagation of the excitation process under various pathologic conditions. Work should be done on the study of the endocardiac potentials and comparison with given epicardial and general electrocardiograms, using new methods and equipment for studying the electrical forces of the heart. Work should be done on comparison of electrophysiologic data with data from biochemistry and biophysics of the myocardium. Useful from

the scientific point of view is the method consisting of patterning the electric activity of the heart (I. T. Akulinichev et al., 1960) for the purpose of studying the various disruptions in the rhythm of the heart or in the shapes of the electrocardiograms.

This method should not be intended for studying the contractile function of the heart. Other methods of synthetic electrocardiology can be used very well for studying this function.

CHAPTER II

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VECTORCARDIOGRAPHY

I. INTRODUCTION

Already in 1913, Einthoven, Fahr and Dewaart actually used the vectorial method for analyzing the electromotive forces of the heart. They proposed to determine the electric axis of the heart with the aid of an equilateral triangle. Subsequently the vectorial concept was given definitive development by the works of Fahr, and in particular by Mann who in 1920 described a method for obtaining a sole curve - i.e. a monocardigram - from the moment axes of the electrocardiogram in standard leads. Mann's method, however, was not broadly disseminated because of the fact that the drawing of a monocardigram from the data of the standard leads involved laborious graphical work. For the purpose of simplifying the monocardigraphic technique, Mann subsequently (in 1938) proposed a special apparatus with a tri-polar galvanometer which made it possible to directly obtain a curve on the film.

A broad and genuine development of vectorcardiography began after 1936-1938 when Schellong et al. (1936, 1937) Hollmann and Hollmann, (1937, 1938) Wilson and Johnston, (1938) proposed, independently of one another, to record a vectorcardiogram with the aid of an electron beam tube. With regard to the further development of theoretical and practical problems of vectorcardiography, the works of Duchosal and Sulzer, (1949) Burch, Abildskov and Cronvich, (1953) Grishman and Scherlis, (1952) and other authors were of great value.

In the Soviet Union the vectorcardiographic method was given further development, thanks to the elaboration by I. T. Akulinichev in 1950 of a homemade vectorcardioscope with an original method of recording. In the course of the last few years there have appeared in the scientific literature interesting works of a number of Soviet authors who devoted themselves to important problems concerning the theory and practice of electrocardiography.

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II. VECTORIAL CONCEPTS IN ELECTROCARDIOLOGY

In contrast to scalar quantities (such as temperature, area, duration, etc.) vectorial quantities (such as force, speed, acceleration, etc.) in addition to magnitude have direction and charge. The geometric sign for a vectorial force is an arrow the length of which indicates its magnitude in the proper units. Its position represents the charge and the direction of the force's activity. Various vectors, occurring during the activity of any force, can have different magnitudes and act in different directions. From this point of view one should understand the following rules.

1. Any vectorial force can be transferred from one place to another without changing its magnitude, direction or charge.

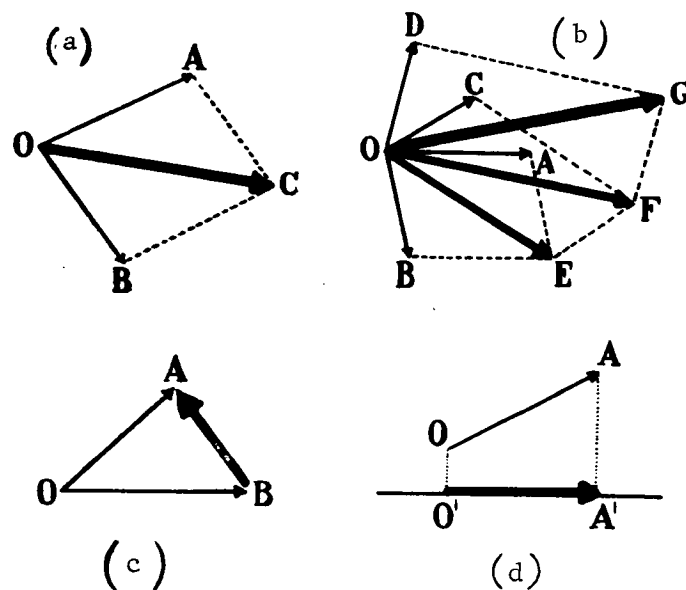


Fig. 56. Vectorial processes. (a) Combination of two vectors— OA and OB, the sum vector—OC; (b) combination of four vectors— OA, OB, OC and OD, sum vector—OG; (c) difference between vectors OA and OB; (d) projection of the OA vector on a horizontal line.

2. In order to combine (add) two vectors arising from a common point, a line is drawn from the tip of each one of the vectors parallel to the other. In this way a parallelogram is formed the diagonal of which (from the common point of origin of the vectors) represents the sum vector of the two vectors in question (Fig. 56a). In the presence of more than two vectors this same method determines the sum vector of any two vectors and hence from the addition of this sum vector and the third vector the resultant vector is determined, and so forth, right up to the obtaining of the last sum /167 vector (Fig. 56b). If the initial vectors do not have a common origin then, in accordance with the first rule, they are transferred just as if they had a sole point of origin.

3. In order to obtain the difference between two vectors having a common point of origin a straight line is drawn between their ends. This line represents the resulting vector (Fig. 56c).

4. In order to project a vector on any line, a perpendicular to the line in question is laid from the ends of the vector to the line in question. (Fig. 56d)

Bioelectric phenomena, which occur during the excitation process of the

heart, spread out along all the surfaces of the area and have magnitude, direction and charge. Based on the theory of the dipole, each moment dipole can be represented as a corresponding vector; the mean magnitude of all of these moment vectors, for example occurring during the depolarization of the ventricles, will represent by itself the mean vector of the heart (AQRS). As stated by Jouve, et al. (1950, 1951), the indicated multiple vectors do not have a common point of origin, but, thanks to an unusual structure of the heart, these diverse initial points are located so near to one another that, for a good approximation, the origin of all of the moment vectors can be considered at a sole point of origin which is called the "electric center" or the "point of zero potential" of the heart. (This point is located in the center of the chest, 2-3 centimeters below the level of lead $V_{1,2}$ or in the center of the sagittal plane, passing through the point of lead V_2 .)

Its location changes little during various phases of cardiac activity.) Thus, if we add the moment vectors V_1 , V_2 , V_3 , V_4 , etc., we can obtain their

mean vector. It is still possible to unite the ends of these moment vectors and to obtain a vectorial loop with a definite configuration which rotates counter-clockwise in the given case (Fig. 57). In this way, by placing the appropriate moment axes on the triaxial system, it is possible to obtain 168 the depolarization complex (QRS) and the repolarization complex (T) of the ventricles and also the excitation of the auricles (P). Mathematically expressed, these loops represent a Lesage figure.

The heart is a three-dimensional organ and therefore all of the specified phenomena (moment vectors, mean vector or vectorcardiogram) should be represented in three dimensions: the horizontal: sagittal and frontal. Consequently, one should not speak of vectorcardiography in general but of dimensional vectorcardiography. In this respect it is terminologically sound and correct to add the letters "sE" (s- from the English word spatial, E- from the words electric vector) to the word "loop". This designation corresponds better to the characteristics of vectorcardiography.

In this way, vectorcardiography is based upon the theory of the dipole and arises from the situation that, as the result of the excitation of all of the individual parts of the heart, an electromotive field develops. (By the word field we mean the given section of the area where any physical magnitude is active.) The vectorcardiographic loop represents a curve depicting the spatial dynamics, i.e. the magnitude and the spatial direction of all of the moment vectors resulting from the excitation process of the heart.

III. TECHNIQUES OF VECTORCARDIOGRAPHY

In this domain, primary use is made of an electron-beam oscilloscope. We do not consider it necessary to describe Mann's monocardigram (1938) since it is not being used at the present time.

The oscilloscope is composed of an electron-beam tube and an amplifier

(Fig. 58). The electron-beam tube produces an electron beam which is concentrated with the aid of special electrodes and falls approximately in the center of the broad circular base (screen) of the tube. The inner surface of the screen is covered by a special substance which, under the effect of the electron-beam, gives off a visible radiation. The radiation has a green or blue color and, thanks to the property of phosphorescence, remains for a certain period of time even after the activity of the electrons has ceased.

The beam of electrons passes through two pairs of plates situated perpendicular to one another. The first pair assumes a vertical position and makes up the horizontal axis of the oscilloscope and the second pair assumes a horizontal position and makes up the vertical axis of the oscilloscope. After great amplification (greater than 200,000 times), the potential from the corresponding leads is connected to the plates referred to above; the potential of one of the leads drives the vertical plate and the potential of the second lead drives the horizontal plate. The electron-beam oscillates vertically along the Y axis each time that a difference develops between the horizontally-placed plates and moves horizontally along the X axis when differences develop between the vertically-placed plates. In this way, depending upon the electromotive force of the heart, the beam on the electron-beam tube moves along the vertical and horizontal axes. /169

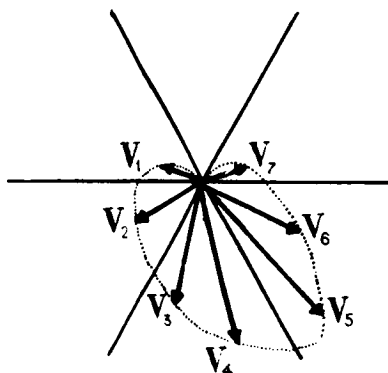


Fig. 57. Derivation of the vertical loop by connecting the ends of the moment vectors V_{1-7} .

Each electron-beam oscilloscope is provided with appropriate devices for obtaining the required amplification in each channel, for determining the speed of the movement of the beam (from 0 to 100 mm/sec.), for developing the beam along the vertical and horizontal axes, and for indicating the times. In addition, for recording the curves from the screen there is a special photographic device, a movie camera or an internal recording system.

It should be noted that the vectorcardioscope can also be used for visual observation of electrocardiograms, phonocardiograms and ballistocar-

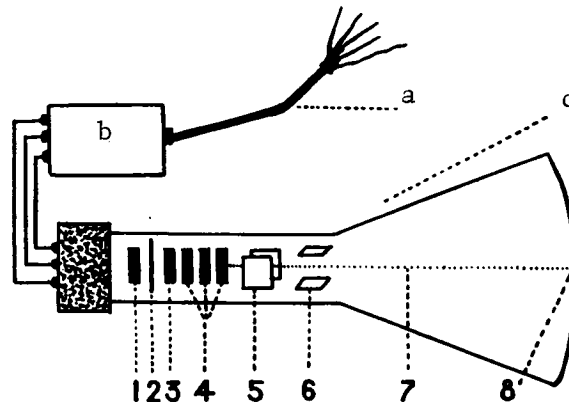


Fig. 58. Schematic of the electron-beam tube. (a) Lead from object being studied; (b) amplifier; (c) electron-beam tube: 1. cathode; 2. exploring electrode, 3. anode; 4. focusing and accelerating electrodes; 5. vertical plates making up the horizontal axis of the oscilloscope; 6. horizontal plates making up the vertical axis of the oscilloscope; 7. electron-beam; 8. fluorescent screen.

diagrams. We concur with Mogilevskiy, Tsukerman and Lapinskiy (1959) that this device can be quite useful for various types of tests when it is used intelligently.

We shall not dwell on such standard details as preparing the skin for attaching the electrodes or the position of the sick person during the time a film is taken. It should, however, be noted that it is better to record the electrocardiogram at the end expiration, during the course of a respiratory pause.

IV. RECORDING SYSTEMS IN VECTORCARDIOGRAPHY

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It is already evident from the description of the electron-beam oscilloscope that a vectorcardiogram can be recorded by attaching to the appropriate plates of the tube two electrocardiographic leads from four points on the body of the person being studied. The polarity of the lead to the oscilloscope is of great importance. In the case of changes in the polarity in one of the two axes of the device, the vectorial loop changes its position in the system of coordinates by 180° . A standard polarity is shown in Fig. 64.

The axes of electrocardiographic leads determine a given spatial plane and, since the heart is a three-dimensional object, it is necessary to select an appropriate combination of leads so as to make it possible to record in a more or less clear form the vertical, sagittal and transverse components of the potentials of the heart. Thus, it is possible to obtain a vectorcardiogram in a horizontal plane by the use of leads which determine the sagittal and transverse components of the electromotive force of the heart; it is

possible to record a sagittal vectorcardiogram by using leads which determine the vertical components of the potentials of the heart; finally, it is possible to record a frontal vectorcardiogram by using leads which determine the vertical and transverse components of the potentials of the heart (Fig. 59).

Many systems for recording vectorcardiograms have been presented which sometimes differ appreciably from one another. All of these systems can be classified under three groups. The first group consists of systems which are based on the equilateral triangle of Einthoven (see page 66); in these systems the electrocardiographic leads are primarily used. The second group consists of those systems in which orthogonal leads are used (the axes of these leads are perpendicular to one another). The third group contains I. T. Akulinichev's system of precordial leads.

The large number and variety of vectorcardiographic systems of recording is explained by the fact that in the make-up of the human organism the chief prerequisites for obtaining a correct vectorial picture of the cardiac forces are absent. The heart is not a geometrically correct figure (it is impossible to represent the heart as a sole dipole having a constant and clear localization, since the heart is situated in a nonhomogeneous volume conductor). From the electrical point of view, there isn't a single point on the human body which can be considered far enough away from the heart so that local potentials of any given part of the heart would not affect the potential of this distant electrode in question. Every author attempts to solve this difficult problem according to his own principle.

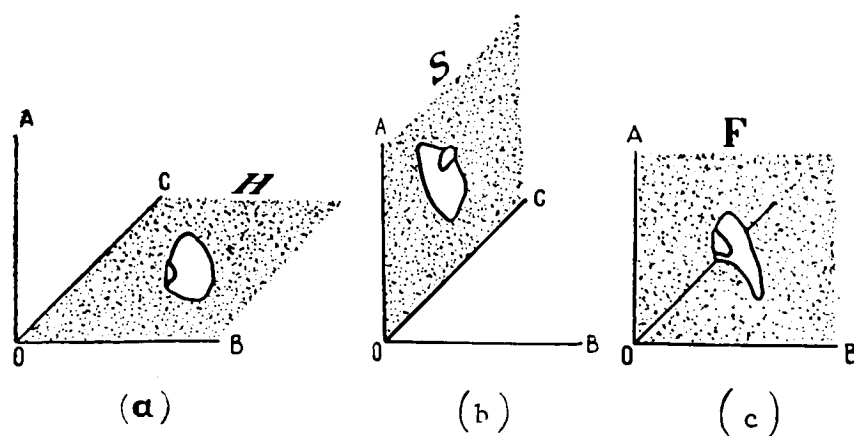


Fig. 59. Formation of a vectorcardiogram on three planes with the aid of leads depicting the vertical (OA), transverse (OB) and sagittal (OC) components of the E.M.F. of the heart. (a) horizontal vectorcardiogram; (b) sagittal vectorcardiogram; (c) frontal vectorcardiogram.

1. Recording Systems Based on the Einthoven Equilateral Triangle

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In 1938 Wilson and Johnston proposed to describe a frontal vectorcardiogram by using lead I for axis X (transverse component) and lead VF for axis Y (vertical component) (Fig. 60). Later on, Goldberger (1954) used this

method, but he used lead aVF and not lead VF for the Y axis. Actually this same principle is also used in describing the "basic" vectorcardiogram with the aid of standard leads I and III (I. T. Akulinichev, 1951) and in describing the frontal vectorcardiogram according to leads II-III and I-II (S. L. Mailyan, 1955).

In 1947 Wilson, Johnston and Kossmann proposed an equilateral tetrahedral system according to which the frontal surface of the Einthoven triangle forms the base of the tetrahedron and its peak coincides with a point which is located on the spine 2 cm. to the left of the seventh thoracic vertebra. Burch, Abil'dskov and Cronvich (1953) find that this system provides satisfactory data since the electrodes are situated at a large distance from the heart in this case. In order to obtain the frontal plane, the electrodes from the right and left hands (lead I) are connected to the vertically-placed plate of the electron-beam tube. The central electrode of Wilson (see page 49) and the electrode from the left leg (lead VF) are connected to the horizontally-situated plate of the tube (Fig. 61a). (In order to obtain a sagittal plane toward the vertically-situated plate the central electrode of Wilson is connected to the last electrode, and to the other pair of plates there is added a lead from the central electrode of Wilson and the electrode from the left leg (lead VF) (Fig. 61b).)

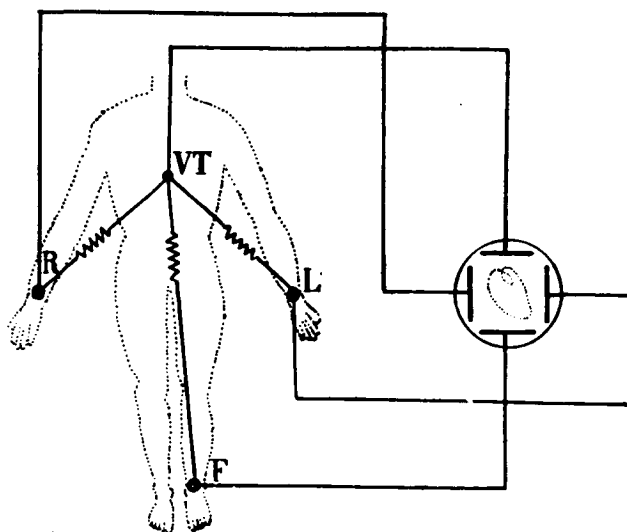


Fig. 60. System for recording the frontal vectorcardiogram according to Wilson and Johnston*

The equilateral tetrahedron method has certain disadvantages and when comparing the curve which is obtained in this way with the data from the

*In all of the figures the oscilloscope with its four plates is represented by a large circle.

model by using a dipole inside an artificial homogeneous male waist, there is a deviation of 15% from the actual picture (Frank, 1954). Thus, it is evident that the Einthoven triangle is not actually equilateral, its plane does not coincide with the frontal plane of the body and the cardiac dipole is not located in the center of the triangle. In addition to this, lead I cannot give a clear picture of the transverse component of the cardiac potentials. It should also be recalled that the axis of the sagittal lead does not always coincide with the sagittal plane of the body and, depending on the constitutional characteristics of the individual and the character of the cardiac disease, it forms a definite angle with the actual sagittal plane.

Burger et al. have offered their system which is actually a modification of the equilateral tetrahedron system. In this system three electrodes are placed on the extremities just as in the case of regular standard leads, and the fourth electrode is placed on the sternum at the level of the axilla.

Donzelot, Milovanovich and Kaufmann, (1950) provide a unique method of recording a spatial vectorcardiogram which is based only in part on the Einthoven triangle. These authors believe that the axis of precordial lead V_6 coincides with the transverse component of the cardiac potentials, the

axis of lead V_2 represents their sagittal components, and the axis of lead

VF coincides with their vertical component. Based on this, the authors propose to record a horizontal vectorcardiogram with the aid of leads V_2 and V_6

and a frontal vectorcardiogram by means of leads V_6 and VF. The chief dis-

advantage of this system lies in the fact that the axes of the leads in question do not always coincide with the corresponding components of the cardiac potentials. Thus, it is evident that the plane formed by leads V_2 and V_6

is not absolutely horizontal and forms a definite angle as regards the actual horizontal plane. In addition to this, points V_2 and V_6 are not at the same

level and are located higher than the points of zero potential of the heart. We no longer speak of the fact that in this system the electrodes are situated rather close to the heart.

Jouve et al. (1950) propose a recording system which is practically the same, but they record the horizontal and sagittal vectorcardiogram by a different method. They determine the points of zero potential of the heart, basing their work on the following electrocardiographic facts described by Duchosal and Sulzer (1949). On the one hand the electrocardiographic curves obtained from the two ends of the straight line, passing through the zero potential point of the heart, have, as regards one another, complexes with a reflected image of all of the components. On the other hand, electrocardiograms with various leads having parallel axes are characterized by a similar picture. In order to determine the horizontal plane which includes the zero potential point of the heart, the authors selected as a base an

electrocardiogram in lead I and sought a similar tracing in the region of the left axillary lines. Jouve considers that the plane of that unipolar lead in the left axillary region, in which the tracing of lead I is recorded, coincides with the actual horizontal plane passing through the center of the zero potential of the heart. For determining the sagittal plane, passing through the zero potential point of the heart, there is first of all determined, according to the principles of the authors which have been referred to, the horizontal plane oV_6-oV_6R (which passes through the zero point) and

then, at the level of the obtained plane, there are determined those points on the front and back surfaces of the chest where there are recorded electrocardiograms with a reflected image of one another. A disadvantage of this method is once more the rather close arrangement of the electrodes. Besides this, this method for determining the electric center of zero potential of the heart should not be considered an accurate method nor easily acceptable in practice.

2. Orthogonal Systems

The first orthogonal (rectangular) system was developed in 1937 by Schellong, Heller and Schwingel. These authors placed the electrodes on the chest in such a way that they obtained orthogonal leads coinciding with the transverse, vertical and sagittal components of the electric field of the heart. In this case the electrical center of the heart was located ahead of and above the center of the axes of these bipolar leads.

Based on this principle, Duchosal and Sulzer (1949) proposed a rectangular system for recording (Fig. 62). The common electrode B (0 according to the authors) is situated in the right lumbar region in line with the medial border of the scapula. Electrode A is situated in the region of the spinous process of the right scapula, electrode C is situated in the left lumbar region at the level of electrode B, and electrode D is at the same level near the anterior axillary line. In this way three bipolar leads are obtained and the axis of lead (C-B) coincides with the transverse component of the electric field of the heart, the axis of lead B-A corresponds to its vertical component, and the axis of lead D-B represents its sagittal component. Consequently, by using leads C-B and D-B it is possible to record a horizontal vectorcardiogram, by using leads B-A and D-B it is possible to record a sagittal vectorcardiogram, and with the aid of leads B-A and C-B it is possible to obtain a frontal vectorcardiogram. /175

An advantage of this system lies in the fact that the electrodes are situated at an equal distance from the heart and are sufficiently far, electrically speaking, from the dipole of the heart, acting in the center of the given geometric figure. It is, however, necessary to keep in mind that the vertical axis of this system is twice as large as its sagittal or horizontal axis and this may result in inaccuracies since, in the joining of the center of the rectangle with the corresponding peaks, dissimilar central angles are formed (Grishman, 1954).

Grishman, Borun and Jaffe, (1951), found that if, instead of taking a

rectangular figure we take a cubic geometrical figure, the disadvantage in the Duchosal and Sulzer method is reduced. They consider that the electric center of the heart, E, is located in the center of a definite sphere and by means of eight separate points on the surface of the given sphere it is possible to set up a cubic figure. These points are located at an equal distance from E and thus the central angles must be the same (Fig. 63a).

The localization of the electrodes in the cubic system is the same (Fig. 63b). In position 1 situated in the region of the right posterior axillary line at level II or III of the lumbar vertebrae is found the common electrode for points A-, B- and C+. In position 2 in the region of the right anterior axillary line at the level of the previous position electrode B+ is situated. In position 4, situated at the level of the right spinous process of the scapula on the right posterior axillary line, electrode C is located. In this way, three orthogonal bipolar leads are formed whereby lead AA determines the transverse component of the electric field of the heart, lead CC corresponds to its vertical component and lead BB represents its sagittal component. Thus, in the corresponding connection of these leads to the plates of the oscilloscope (according to the normally used standard of polarity) it is possible, with the aid of leads AA and BB, to obtain a horizontal vectorcardiogram, with the aid of leads CC and BB to obtain a sagittal vectorcardiogram, and with the aid of leads CC and AA to obtain a frontal vectorcardiogram.

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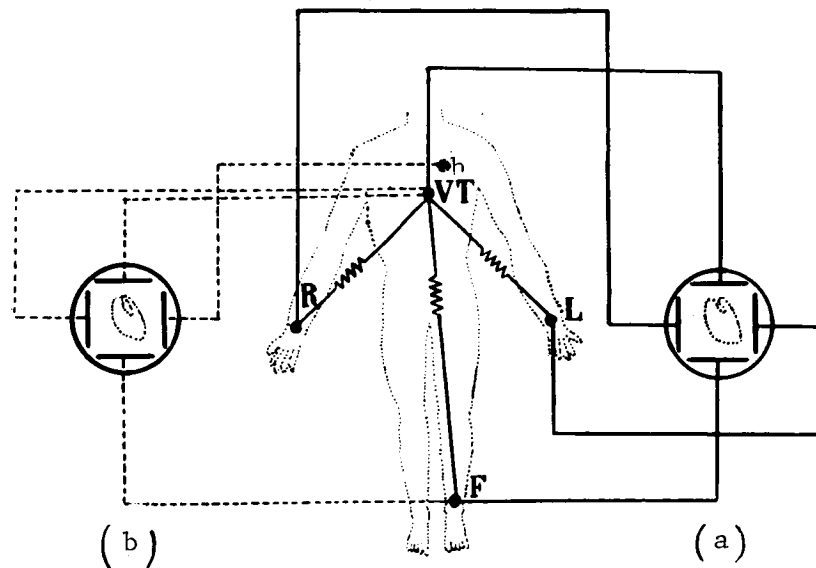


Fig. 61. Equilateral tetrahedron system: (a) recording of the frontal vectorcardiogram; (b) recording of the sagittal vectorcardiogram; (h = last electrode.)

The cube method undoubtedly has definite advantages. Here the electrodes are situated at a fairly large distance from the dipole of the heart and we

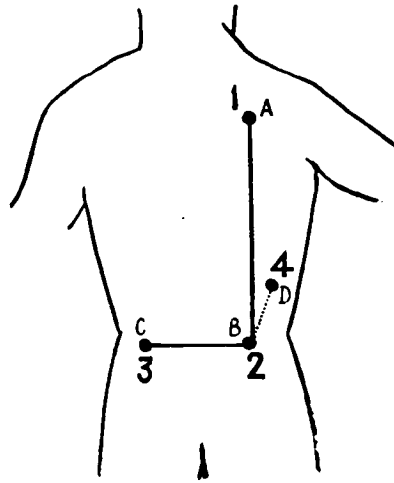
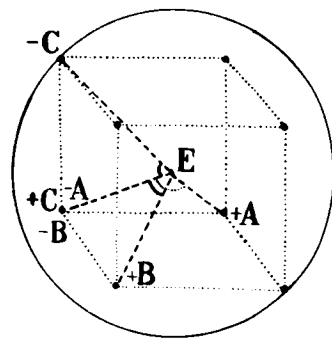
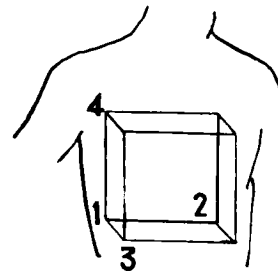


Fig. 62. Arrangement of the electrodes in the Duchosal and Sulzer orthogonal system (explanation in the text).



(a)



(b)

Fig. 63. The cubic recording system according to Grishman and Scherlis. (a) Geometric figure of a cube, the electrical center of the heart (E) is located in the center, the inner angles are equal; (b) arrangement of the electrodes (explanation in the text).

have to deal with a rectilinear and symmetric geometrical figure. Johnston (1961) finds that it is not possible to determine, with a great degree of accuracy, the transverse, vertical and sagittal components of the electrical field of the heart. He proposes that lead VF or aVF be recorded for the vertical component or else a lead of such a type that one electrode is placed on the left leg and the second in the region of the neck. We believe that the cube method which has been worked out well by Grishman and Scherlis (1952) is appropriate for practical work. It does not present any technical

difficulties and can give a sufficiently accurate picture of the components which are needed for a spatial vectorcardiogram.

In scientific literature a whole group of other orthogonal systems have been presented. We can only discuss a few of them.

Schmitt and Simonson, (1955), propose so-called SVECII and SVECIII systems for stereovectorelectrocardiographic observation (SVEC stands for stereovectorelectrocardiography). In the case of the SVECIII system three electrically orthogonal leads are formed. For this purpose the authors used 14 positions of the electrodes, four of which were for the transverse lead, eight for the sagittal lead and two for the vertical lead. These authors /177 found that this system has definite advantages over other orthogonal systems.

Blackburn and Simonson (1957), use three anatomically orthogonal leads. In this system the horizontal lead is determined by placing the electrodes in the right and left mid-axillary lines at the level of the fifth vertebra; for the purpose of obtaining the vertical lead one electrode is placed at the level of the determined horizontal plane in the region of the left peristernal (around the breast-bone) line, and the second electrode is placed in the region of the forehead; for obtaining the sagittal lead the electrodes are placed on the same horizontal plane, one of the electrodes on the right peristernal line, and the second electrode opposite the first one in the region of the right scapula. These authors consider this system to be very valuable and with the aid of this method they determine with a great degree of accuracy the common duration of the QRS complex.

Helm, (1957), describes three orthogonal leads. He uses two large electrodes from a sponge which has been wetted with a physiologic solution and five small ordinary electrodes. The author finds that, by using this system, relatively similar vectorial characteristics are obtained for all parts of the heart.

In his system of recording for obtaining the X, Y and Z components of the electromotive forces of the heart, Frank (1954, 1956) uses seven electrodes: five of these electrodes in the region of the anterior surface of the chest, one on the left leg and one on the right side of the neck. Mac Fee (according to Burger, van Brummelen and van Herpen, 1961) uses nine electrodes: seven of them on the chest, one on the left leg and one on the right side of the head. In the Burger system (1961) there are five electrode positions: two on the chest, one lead each on the right and left shoulders and one on the lower left limb. These systems differ from the previous ones in that they are not homogeneous but are heterogeneous and the limbs are used in these systems for attaching the electrodes.

3. Precordial Leads System

In the elaboration of the principle of recording vectorcardiograms great value is attributed to the works of I. T. Akulinichev, to his theoretical and practical experiments. The various vectorcardioscopes which he has constructed are presently being widely used in the Soviet Union.

In this system of recording, the electrodes are placed at five positions on the chest (Fig. 64). Electrode 1 is placed on level with vertebra II, to the right of the sternum; electrode 2 is placed on the left subclavicular region; electrode 3 on vertebra V along the left mid-clavicular line; electrode 4 to the right of the epigastric corner and electrode 5 from behind, between the corner of the left scapula and the spinal column. M. B. Tartakovskiy (1960) proposes to use for the fifth position the last point of "Neb" which is situated on vertebra V on the posterior axillary line. Zh. /178 A. Teslenko (1958) finds that the use of "Neb's" lead gives valuable results, especially if at the same time the electrocardiogram is recorded with the Neb lead.

By using the positions which have been referred to above it is possible to obtain a number of bipolar leads from which two leads (1-3 and 2-4) coincide with the anterior surface of the body and have orthogonal axes, and the other leads (5-1, 5-2, 5-3 and 5-4) pass through the entire thickness (mass) of the chest and surround the heart from all sides. By properly combining these leads we can study the electrical field of the heart in five projections (Fig. 64).

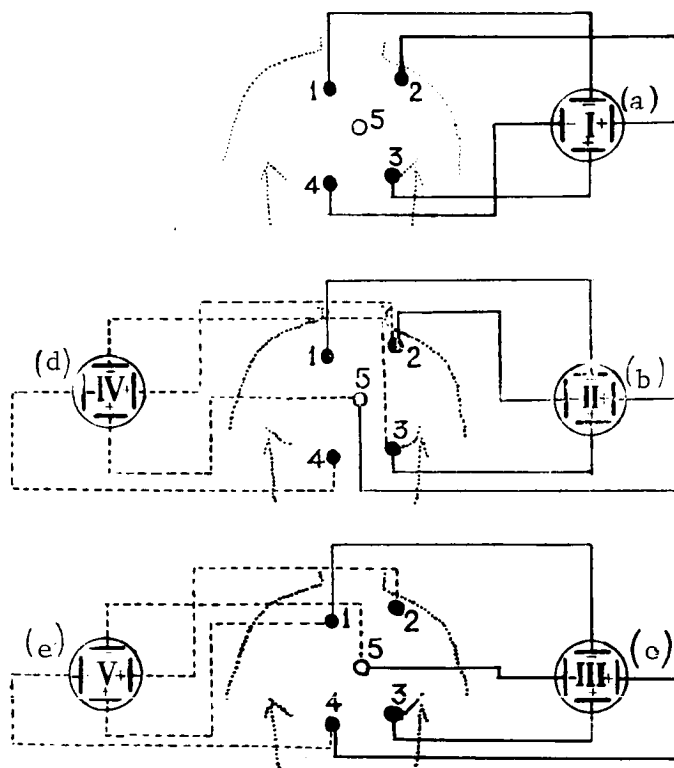


Fig. 64. A precordial recording system according to I. T. Akulinichev. The arrangement of the electrodes, the polarity of (a) projection I, (b) projection II, (c) projection III, (d) projection IV, (e) projection V.

The first projection is obtained when lead 1-3 is placed on the vertical axis of the oscilloscope and lead 4-2 is connected to its horizontal axis (Fig. 64a). This projection coincides with the plane of the anterior surface of the heart and primarily reacts on the currents of the front wall of the heart which can be directed upwards, downwards, to the right or to the left (as regards the view from the front). /179

In order to obtain the second projection, lead 1-3 is connected to the vertical axis of the oscilloscope and lead 2-5 to the horizontal axis of the oscilloscope (Fig. 64b). This projection coincides with the plane which passes through the region of the left ventricle and reacts primarily on the currents of the left side wall of the heart which can be directed forwards, upwards, to the left, and backwards (as regards the view from the left shoulder region somewhat at the rear).

The third projection is formed when lead 1-3 is placed on the vertical axis of the device and lead 5-4 is placed on the horizontal axis (Fig. 64c). This projection coincides with the plane passing through the region of the right ventricle and reacts on the currents of the right anterior-posterior part of the heart which can be directed forwards, downwards, to the right, and backwards (as regards the view from the right lower diaphragm region).

To form the fourth projection, lead 4-2 is connected to the horizontal axis and lead 3-5 is connected to the vertical axis of the oscilloscope (Fig. 64d). This projection coincides with the plane of the region of the apex and rear surface of the left ventricle and reacts on the currents of the left posterior diaphragmatic surface of the heart which can be directed downwards to the right, downwards to the left and upwards to the left posteriorly (as regards the view from the side of the lower diaphragm region).

The fifth projection is formed by connecting lead 4-2 to the horizontal axis of the device and lead 5-1 to the vertical axis (Fig. 64e). This projection coincides with the plane of the base of the heart and reacts on the electrical forces of the heart which can be directed downwards, forwards to the right, upwards to the left and backwards (as regards the view from the side of the right shoulder).

As is evident from what has been described above, in leads I, II and III on the vertical axis of the oscilloscope there falls the potential of one and the same lead 1-3, in projections I, IV and V on the horizontal axis of the oscilloscope there falls lead 4-2 which is more common to them. From this it is possible to come to the conclusion that the differences in deviations along the horizontal axis in projections I, II and III depend only on the voltage of the horizontal axis of the oscilloscope, i.e. on the potential of leads 4-2, 2-5 and 5-4 respectively, and the differences in the deviations on the vertical axis in leads I, IV and V depend upon the voltage of the vertical axis of the oscilloscope, i.e. upon the potential of leads 1-3, 3-5 and 5-1 respectively. Using these positions, M. I. Reiderman (1961) shows that the deviation along the X and Y axes have the following regularity:

$$X_I + X_{II} + X_{III} = 0 \quad \text{or} \quad X_I + X_{II} = -X_{III},$$

$$Y_I + Y_{IV} + Y_V = 0 \quad \text{or} \quad Y_I + Y_{IV} = -Y_V.$$

From here the author comes to a number of interesting conclusions which /180 have a practical value in ascertaining the accuracy of the method of taking vectorcardiograms. Thus, in leads I, II and III the arrangement of the loops as regards axis X must be the same, and in leads I, IV and V the loops must have an analogous arrangement as regards axis Y. Or else, if in one of the first three leads the loops do not have any width at all and represent one line, then there must be a reflected image of the loops in the other two leads since, according to the formula of M. I. Reiderman, if we allow that X_{II} is equal to 0, then X_I is equal to $-X_{III}$.

The precordial lead system has certain disadvantages. The fact is, we are not dealing with a correct geometric figure and out of all of the five leads with a determined spatial plane (frontal) only the first lead coincides. The others do not represent planes and, as I. T. Akulinichev writes (1960), they are hypothetical. This situation makes it impossible to compare the obtained curves with the data of the regular electrocardiographic leads (the supposition of M. B. Tartakovskiy, 1959) concerning the use of the last point of Neb. To compare the obtained vectorcardiogram with the electrocardiographic data, we need to record at the same time the bipolar chest leads of Neb (1938) which, in regular practice, are hardly ever used. In addition, this situation hampers the basic task of vectorcardiography - that of drawing up a spatial representation of the electric forces of the heart. With this purpose in mind, I. T. Akulinichev (1960) uses a relief vectorcardioscope together with a three-channel device with which on only one lead is there obtained the spatial representation of a vectorcardiogram. In this case the direction of the curve forwards or backwards is determined by the intensity of the beam: in the case of a backward movement of the loop a darkening of the tracing of the beam is obtained, and in the case of a forward movement of the loop, the intensity of the beam becomes lighter. This method, however, cannot give an accurate representation of the spatial arrangement of the vectorcardiogram and, furthermore, it presents technical difficulties. I. T. Akulinichev (1960) proposes a three-dimensional precordial system with the use of three leads: a longitudinal lead where the electrodes are placed in the region of the right forearm and at the fifth chest position, a transverse lead with the electrodes in the region of the forearm and to the right of the bladder and appendix, and an anterior-posterior lead with electrodes on the spine and on the second chest position. However, the author himself states that it is impossible to guarantee the possibility of the five projections.

In I. T. Akulinichev's system of precordial leads, a figure is created which resembles a quadrangular pyramid with the base applied to the anterior chest wall and with the apex applied to the spine (Fig. 65). An analogy of /181 this type is only approximate because, as we have already stated, except for the first projection, the other projections do not represent any definite plane whatsoever. In addition to this, even if there is a correct geometric figure, under the conditions of the human body, and in particular the chest,

this figure would be subjected to distinct changes since the electrical characteristics of the various tissues are not the same.

It should also be noted that in this system the electrodes are located rather close to the heart and this can result in definite distortions - first of all because in proportion to the proximity of the electrode to the surface of the heart there is an increase in the effect of the local potentials of the individual parts of the heart on the corresponding electrode and a harmonious picture of the total electric field of the heart is not obtained. Secondly, as noted by Jouve, Sene and Pierron (1954), when the electrodes are situated close, the corresponding leads can be so short that they are not in a position to record the sharply dispersed electrical phenomena.

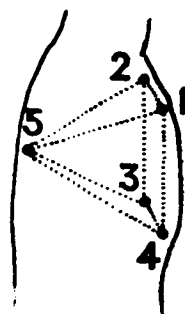


Fig. 65. The precordial system of I. T. Akylinichev resembles the geometric figure of a quadrangular pyramid.

However, despite all of this, the precordial leads have certain advantages over other systems. Under conditions where the electrodes are close to the heart, the technical necessity of having a large amplification of the live currents of the heart is eliminated since the amplitude obtained under these conditions is great enough for a close reading of it. On the other hand, the axes which are used in this system of leads pass through nearly similar tissue and the significance of the various tissue characteristics is, fortunately, brought to a minimum. The experience of Soviet authors shows that the precordial system of I. T. Akulinichev gives thorough and detailed information on the electric activity of the heart and, when the obtained data are correctly analyzed, this system truly offers distinct advantages over other vectorcardiographic systems. However, we should not agree with the attempts of some authors to prove that the chief advantage of this system lies in the fact that it makes it possible to study a vectorcardiogram from the topographic point of view. Theoretically speaking, this point of view is not competent since, as a result of the excitation of the heart, an entire electric field is created and any lead records the total picture of the potentials and not just the local potentials of the portion of the heart corresponding to its location.

Besides the above-mentioned principles of recording, there is also a

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stereovectorcardiographic principle the aim of which is to obtain, on the basis of only a single loop, a representation of the spatial arrangement of the vectorcardiogram. A number of authors propose to record two vectorcar- /182
diograms from the determined planes of the chest and to observe them through special glasses which produce a spatial impression. Burch, Abil'dskov and Cronvich (1953) presented a system of an equilateral tetrahedron with the inclusion of a certain modification in the circuit. A stereoscopic picture can also be obtained with the aid of repeated photographing of the pattern of the vectorial loop. It should be noted that at the present time stereovectorcardiographic methods are not being widely used.

Thus, at the present time there are a large number of various principles and systems for recording vectorcardiograms. Each one of these systems, besides having certain advantages, also has a number of disadvantages, even though all of these disadvantages have to do with an incorrect geometric figure which is excentrically located in the heterogeneous volume conductor. The importance of each method should be evaluated starting from the theoretical premises on which the method is based, and continuing through to the simplicity and dependability of the recording technique. In our practical work we predominantly use the precordial lead system of I. T. Akulinichev.

Comparing the pictures which have been obtained utilizing eight different methods of orthogonal leads of vectorcardiograms, Simonson, Schmitt and Nakagawa (1959) detected a great difference between the data of these systems. Langner et al. (1958), in comparing vectorcardiograms with eight orthogonal systems of leads, arrives at the opposite conclusion. Kossmann (1958) also did not find any great difference between the pictures of spatial vectorcardiograms obtained by means of various recording systems. According to our opinion, one thing is evident: that such a variety of recording systems cannot serve as a sound basis for the development of this new and complicated field. It is necessary to achieve a universal standardization of the methods and for this purpose, as indicated by Burger, van Brummelen and van Terpen (1961), it is necessary to carry out a thorough investigation of the electrical field in the chest and its distribution along the surface of the body.

V. NORMAL VECTORCARDIOGRAMS

On a normal vectorcardiogram three deflections are differentiated and each one has an initial part, a body and a final part; in addition to a common zero point where the deflection starts and finishes (Fig. 66). The large external deflection corresponds to the process of depolarization of the ventricles and is referred to as the QRS complex. Inside of this deflection is found a small deflection T which indicates the process of repolarization of the ventricles. The smallest deflection, P, represents the auricular excitation process and very often it is not seen since it overlaps on the initial and final part of the QRS and T loops, especially /183
in the case of a photograph of the curve. The P wave can be detected with the aid of a movie camera or when there is a great amplification of the currents brought about by connecting the attachment block to the regular device

(V. F. Sysoyev 1958), or by using a differential vectorcardiogram when the rays become much brighter on the two-channel oscilloscope at the time that the P wave is recorded (Sano, Hellerstein and Vayda, 1957).

The QRS complex, which is the principal component on the vectorcardiogram, is formed in the following manner. At the first instance of ventricular excitation, the vector of the electrical forces is directed anteriorly to the right and the initial part of the complex which corresponds to wave Q of the electrocardiogram deviates anteriorly to the right. When the excitation spreads further along the diaphragm and ventricles, the electric vector moves posteriorly since the excitation process primarily spreads to the rear in the left ventricle. In this case the QRS complex moves posteriorly, downwards and to the left, forming the apex of the complex corresponding to the R wave of the electrocardiogram. When the base of the heart is excited, the vector of electric forces is oriented anteriorly and upwards and the QRS, with its end part corresponding to the S wave of the electrocardiogram, moves upwards and forwards and terminates in its starting point.

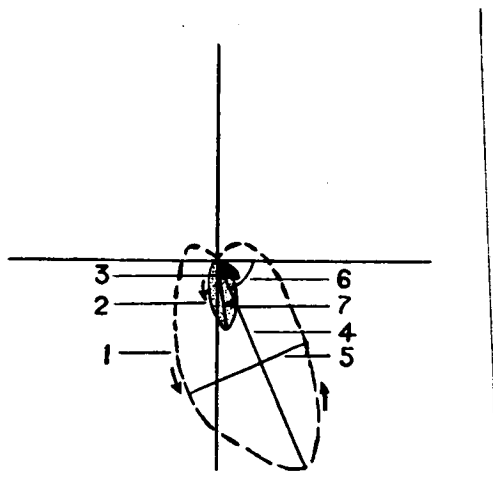


Fig. 66. A normal vectorcardiogram (schematically): (1) the QRS complex; (2) the T wave; (3) the P wave; (4) the maximal vector of the QRS complex; (5) the maximal width of the QRS complex; (6) the angle determining the location of the QRS complex in the rectangular-coordinate system; (7) angle of deviation of QRS-T; the arrows indicate the direction of the tracings counter-clockwise; the time marks at the beginning and end of the tracing are relatively thicker and closer to one another than in the case of the other parts of the tracing.

In describing the characteristics of the tracing the following indicators of the tracing should be given (Fig. 66).

1. The form of the tracing may be diverse: it can have a leaf-shaped, ellipse-shaped, an indeterminate form, or the form of a figure eight, of a sphere, and so on. M. B. Tartakovskiy (1959) proposed to determine the form

of the tracing not by its similarity to various other figures but by certain criteria which make it possible for him to differentiate two basic types of tracings: the first type has one axis inside the tracing and the tangents and the points of maximum deviation on the horizontal and vertical line (see below) do not bisect the tracing; the second type can have several axes; the axis and the tangents can bisect the tracing. Attention /184 should be paid to the contour and also to the so-called forces of the initial part, the body and the final part of the loop. According to the data of Young, Wolff and Chatfield, (1956), the initial forces cause a deviation of the QRS loop to the right and/or upwards from the zero point and the corresponding start of the Q wave in the electrocardiogram of leads I, aVF and V₆;

the final forces cause a deviation of the end of the QRS loop and indicate the start of the S waves in these electrocardiographic leads.

2. The maximum vector of the loop represents a line connecting the zero point to the most distant point on the tracing of the loop. Its magnitude is expressed in millivolts (1 mv=10 mm). The maximum vector actually coincides with the principal or maximum axis which, in the majority of cases, differs only slightly from the mean vector of the heart. Some authors (I. I. Bykov, 1958; M. B. Tartakovskiy, 1959) determined the maximum axis by the following method: placing the loop on the rectangular-coordinates system, they determined the maximal deviation of the loop along two axes of the system, i.e., along the horizontal and along the vertical. They then drew tangents through these points (parallel to the axes of the system) and, connecting the point of intersection of these two tangents to the center of the system, they obtained the maximum axis. We believe that this method can be of value when there is not one but two or more maximal vectors.

3. The maximum width is the expression in millivolts of the widest part of the loop. In carrying out the measurements, the axis of a given distance must be perpendicular to the maximum vector of the loop.

4. The area of the loop is determined by a planimetric method. The magnitude of the maximum vector and the maximum width already provide an approximation as to the size of the area of the loop since the area is actually determined according to the amplitude of the maximum deviations along axes X and Y. However, as noted by M. B. Tartakovskiy, (1959), still another factor plays an important role - the factor of the conformity in the rates of growth of the potentials along the two axes. The area of the loop is that much greater in proportion to the difference in the rate. In other words, the area of the loop actually depends upon the asynchronism between the electric phenomena in the two axes (I. I. Bykov, 1958).

5. The direction of the ray or the tracing is determined by comparison with the movement of the needle and is shown by an arrow. The direction depends upon the picture of the electric phenomena and upon the speed of their formation on the X and Y axes. If the loop has a twist then the recording of the ray below this part is carried out in the opposite direction. Twists of this type were referred to in 1959 by M. B. Tartakovskiy as true nodes in contrast to false nodes where the direction of the ray does not change since /185

there does not take place a bisecting of the loop but rather a sharp drawing together or osculation of two of its parts.

6. The rate of formation of the loop is determined with the aid of a rate recording system which, in accordance with the development of the device, establishes, according to definite intervals, the individual parts of the darkening along the tracing of the loop. In the case of a rapid movement of the loop these darkenings become arranged at a certain distance from one another. In the case of a slow movement of the loop they are arranged close to one another and the tracing of the loop between them is relatively thicker. The rate of formation of the loop depends upon the rate of spread of the excitation process.

7. The direction of the loop is determined with the aid of a rectangular-coordinate system. The zero point of the loop is located at the center of the coordinate system and the deviation from the X axis is determined. In scientific literature there are various proposals concerning the polarity of the coordinates. The most widely used proposal (the one which we also use) is the following system (Fig. 67): the lower semicircle is positive, the upper semicircle is negative. The reading of the angles starts from the left side of the X axis and follows the path of the needle (clockwise) in the lower semicircle, against the path of the needle (counterclockwise) in the upper semicircle. The right side of the X axis is designated as $\pm 180^\circ$, the lower side of the Y axis corresponds to $+90^\circ$, and the upper side of the Y axis corresponds to -90° . Four quadrants are obtained: the lower left quadrant or I, the upper left quadrant or II, the upper right quadrant or III, and the lower right quadrant or IV. For the precordial system of I. T. Akulinichev (1960) the following principle for determining the polarity of the coordinates: for 0° the positive pole of the horizontal axis of the oscilloscope is used, and for $+90^\circ$ the positive pole of the vertical axis is used. This principle conforms to the universal system of polarity.

In scientific literature there are still other methods for determining the polarity of systems of coordinates. M. I. Kechker (1960), Ye. A. Kyandzhuntseva and V. I. Makolkin (1958) proposed to designate the upper side of the Y axis for leads IV and V as $+90^\circ$ and to designate the lower side as -90° . M. B. Tartakovskiy (1959) proceeds from the polarity of the electrodes and (+) along the X axis is designated as 0° and (+) along the Y axis is designated as $+90^\circ$. The regularly used system is kept for leads II and III. /186 For lead I the polarity of the X axis is changed, and for leads IV and V the polarity of both the X axis and the Y axis is changed. V. I. Makolkin (1960) proposed in lead III to place 0° on the negative pole of the X axis for the purpose of allowing this system to present a picture of the fact that in the rectangular-coordinate system the lower right quadrant is positive.

8. The angle of deviation between the maximum vectors of the QRS and T waves is determined. It should be kept in mind that in warm-blooded animals and in man the processes of depolarization and repolarization of the ventricles do not take place in the same manner and therefore loops QRS and T have different directions.

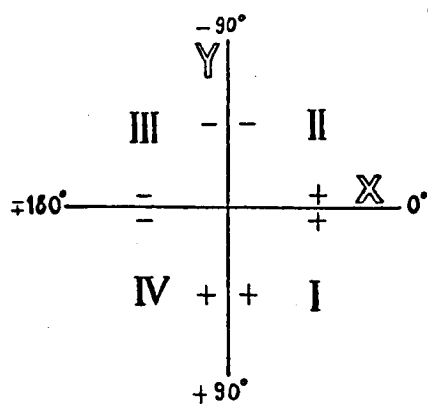


Fig. 67. A regularly used system of coordinates. The designation of the quadrants is done according to Helm's principle: I - positive value along the X and Y axes; II - positive value along the X axis, negative value along the Y axis; III - negative value along the X axis and the Y axis; IV - negative value along the X axis, positive value along the Y axis.

9. The spatial arrangement of the vectorial loop can be easily determined with the cubic system of recording, since on the frontal plane the movement of the loop upward-downward and right-to-left is determined; on the sagittal plane a movement forward-backward and upward-downward is observed and on the horizontal plane there is a movement forward-backward and right-to-left. Helm (1956), based on the proposition of Robertson (1957), that the movement of the vector in the direction of the researcher should be considered positive, recommends a system of coordinates in which the survey of the frontal plane is carried out in front, the survey of the sagittal plane is carried out from the left, and the horizontal plane from below. V. F. Sysoyev (1960), also recommends that the sagittal plane be examined from the left side of the subject being studied in order to have a more convenient spatial picture of the vectorcardiogram. It is well known that Grishman and Scherlis, (1952) carried out the survey of the sagittal plane from the right side of the subject being studied.

In the precordial recording system, the principal difficulty in determining the spatial arrangement of the vectorcardiograph is linked with the fact that there is no definite sagittal component which would make it possible to determine the deviation of the loop forwards and backwards. In order to facilitate the spatial analysis of precordial vectorcardiograms, I. T. Akulinichev, (1960), proposed to mount the leads which have been obtained by means of the so-called unfolded envelope method (Fig. 68). A large rectangular-coordinate system is selected and lead I is placed in the center so that segment 4-2 coincides with axis X and segment 1-3 coincides with axis Y. The other leads are placed in such a way that the centers of leads II and III are located on the X axis, and the centers of leads IV and V are located on the Y axis. Then the entire system is rotated 45° counterclockwise so that the X axis coincides with the axis of segment 4-2 and the Y axis coincides with the axis of segment 1-3. It should be noted that for

lead I it may not be necessary to rotate the rectangular-coordinate system by 45° because this lead practically coincides with the existing frontal plane of the body, on which the electric axis of the heart is determined.

I. T. Akulinichev (1960) found that the method of the unfolded envelope /187 makes it possible to obtain a spatial evaluation of the electric field of the heart since, in this method, the spatial arrangement of the loops can be determined by proceeding from the direction of the loop according to the points of location of the electrodes on the chest. He finds two basic advantages in this method. First, a mounting of this type makes it possible to verify the accuracy of the picture-taking method: in the case of a correct recording the loops must have a similar maximum distance from the zero point on the Y axis in leads I, II, III and on the X axis in leads I, IV, V (in leads I, II, III the common segment 1-3 is connected to the Y axis and in leads I, IV, V the common segment 4-2 is connected to the X axis). Secondly, if the envelope is folded in half it is possible to obtain an approximate model of a spatial vectorcardiogram of a precordial system.

A number of authors (M. I. Kechker, 1960; V. I. Makolkin, 1960) propose an original principle for determining the sagittal component in a precordial vectorcardiogram. It is evident that in leads II and III the same 1-3 segment is connected to the Y axis. Thus, the distinctive characteristic of a /188 vectorcardiogram in these leads depends upon that segment which is connected to the X axis, i.e. upon segment 2-5 for lead II and upon segment 5-4 for lead III. Hence, when recording lead II, electrode 2 can be transferred in the direction of electrode 4; and when recording lead III electrode 4 can be moved in the direction of electrode 2 and a determination can be made of that point on the anterior surface of the chest where the same picture is obtained in leads II and III. It can be thought that this segment (detected point-5) corresponds to the sagittal component. Such a summing up is also made with respect to leads IV and V. In carrying out a more detailed investigation into these problems, M. I. Kechker (1960) established that if with the aid of points 1-3-5 and 2-4-5 three isosceles triangles are formed then the perpendicular from point-5 falls on the point of intersection of the bases of these triangles. Based on this, we come to the conclusion that the sagittal component for a precordial system corresponds to the segment which is formed by connecting point-5 with the point of intersection of segments 1-3 and 4-2.

For practical work and rapid determination of the spatial arrangement of the QRS complex directly with the screen, it is possible to proceed from the following schematic which has been set up by M. I. Reiderman (1961) based on the data of a number of authors (Fig. 69).

1. The deviations upward and downward come out well in leads I, II and III since the potential of the vertical axis is common to them. /189
2. The deviations to the right and to the left come out well in leads I, IV and V since the potential of the horizontal axis is common to them.
3. In the case of predominance of the electric forces to the left and backward, the loop moves to the left in lead I, to the right in lead III, and

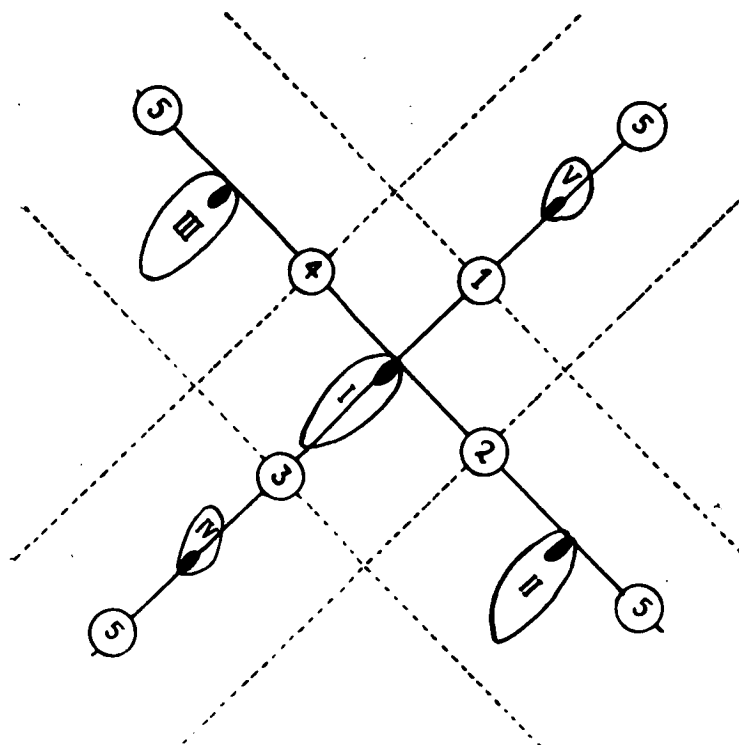


Fig. 68. Arrangement of a precordial vectorcardiogram according to the unfolded envelope method (explanation in the text).

in lead II the loop does not undergo any significant changes; the reverse is observed in the case of the presence of forces with a direction to the right and forward.

4. In the case of changes in the electric axes to the anterior-posterior direction, the QRS loop displaces itself into leads II and III; in lead I the loop does not undergo any significant changes.

Thus, we carried out an analysis of the vectorcardiogram under the criteria which have been referred to and determined the spatial arrangement. We do not construct wire patterns, as some authors have proposed, and we do not carry out a stereoscopic plotting concerning the method which has already been discussed.

It should be noted that if the spatial arrangement of the vectorcardiogram does not nearly depend upon which system of recording is used to determine it, then the various leads or planes of the individual systems play a specific role. From this point of view it is necessary to know the characteristic of the vectorcardiogram which is obtained with the aid of two principal systems of recording, the precordial system and the cubic system.

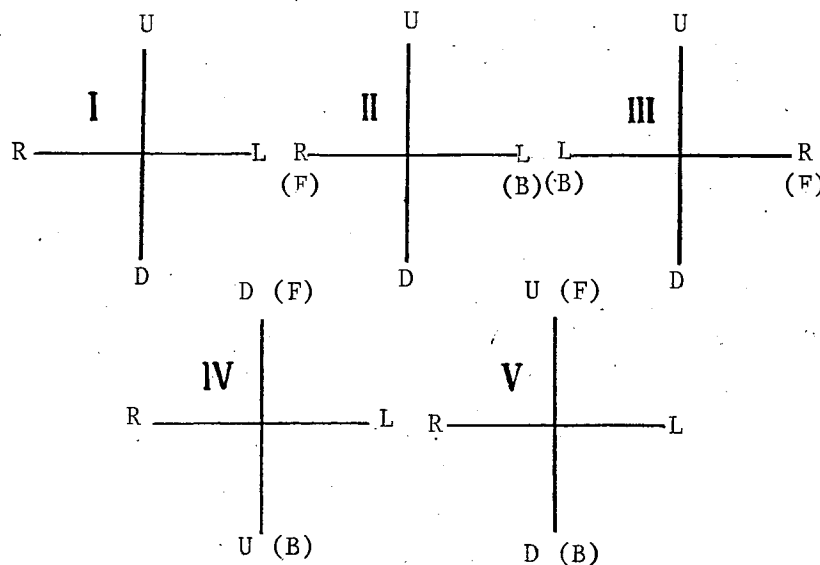


Fig. 69. Schematic for determining the deviation of the vectorial loops directly by means of the M. I. Reiderman screen. The deviations are studied with reference to longitudinal axis of the heart which is assumed to be in a vertical position. The symbols stand for: U - upwards, D - downwards, R - to the right, L - to the left, F - forward, B - backwards.

1. The Normal Vectorcardiogram in Various Precordial Leads (Fig. 70)

The QRS loop can have a leaf-shaped, oval-shaped, ellipse-shaped or spindle-shaped form. Z. Z. Dorofeyeva and I. F. Ignat'yeva (1958) noted that in all of the planes that the loop has a leaf-shaped form, the width is pointed, less often rounded off, or has a cross. According to the data of O. V. Aleksandrov (1960) in 50% of the cases the QRS loop has an ellipse-shaped form, in 40% of the cases it has a leaf-shaped form and in 10% of the cases it has a figure-eight form. In the base, loop QRS is somewhat assymetric as a result of a blurting out in the shape of the positive pole of the posterior part of the QRS loop. The movement of the ray at the start and at the end of the QRS loop slows down somewhat. These parts often cross over at a rather small distance in the opposite side (as regards the direction of the maximum vector of the loop). The QRS loop has one maximum vector, the magnitude of which primarily fluctuates between 1.0 mv and 3.0 mv, the width of the loop equals 0.3-1.2 mv, its area is 0.3-1.2 cm². The contours of the loop are even.

Loop T usually has a narrow ellipse-shaped form. Its area is several times smaller than the area of loop QRS. It has even contours. Its maximum vector deviates somewhat from the direction of the maximum vector of the QRS 190 loop, usually on the side of the starting part of the latter. The movement of the ray when recording the T loop usually has the same direction as when

the QRS loop is recorded.

The P loop is small, it is closed or remains somewhat open and moves vertically downwards whereby in the horizontal and frontal planes the tracing of the loop moves counterclockwise, and in the sagittal plane it moves clockwise. (Sano, Hellerstein and Vaid, 1957.) According to the data of V. F. Sysoyev (1958), the P loop has the shape of an ellipse or a rhombus, its contours are uneven and notched, the largest vector is oriented downward to the left and forward, and the surface which is enclosed within the contours of the loop is approximately parallel to the frontal plane.

In lead I the QRS loop is usually narrow and the movement of the ray can be clockwise or counterclockwise. M. I. Kechker and Sh. I. Shurgaya (1958) in 80% of the cases determined that the movement of the ray is clockwise and in 20% of the cases that it is counterclockwise. E. A. Kyandzhunitseva and V. I. Makolkin (1958) noted that the deviation of the tracing clockwise is observable when the heart is in a vertical position; counterclockwise in a horizontal position, and an alternating path, i.e. the loop is in a figure-eight shape, when the heart is in an intermediate position. The maximum vector of the QRS loop is located in the area between $+35^{\circ}$ and $+80^{\circ}$ and when the heart is in a vertical position it is closer to $+80^{\circ}$ and when in a horizontal position it fluctuates around $+35^{\circ}$. The T loop is located within the QRS loop and the angle of deviation between their maximum vectors does not exceed 37° .

The QRS loop is somewhat wider in lead II than in lead I, usually has an ellipse-shaped form and the tracing of the ray is, as a rule, counterclockwise. The maximum vector is located in the area between $+46^{\circ}$ and $+116^{\circ}$. When the heart is in a vertical position it is closer to $+46^{\circ}$ and when the heart is in a horizontal position it fluctuates around $+116^{\circ}$. The T loop is located within the QRS loop and the angle of deviation between their maximum vectors does not exceed 37° .

In lead III the tracing of the ray moves, as a rule, clockwise. The maximum vector is located in the area between $+57^{\circ}$ and $+120^{\circ}$. The T loop is within the QRS loop and the angle of deviation between them does not exceed 37° .

In lead IV the QRS loop is wide, has a varied form, can have the shape of a figure-eight. The tracing of the movement of the ray is variable but more often clockwise. The maximum vector is located within the area between -45° and -135° . When the heart is in a vertical position it is closer to -45° and when the heart is in a horizontal position it fluctuates around -135° . The T loop is located within the QRS loop, sometimes outside of its limits; the angle of deviation between the maximum vectors of these loops /191 does not exceed 37° .

In lead V the QRS loop appears in a wide variety of different forms, it is not wide and often has steep bends, the tracing of the movement of the ray varies in direction but is more often clockwise. The maximum vector is located in the area between -15° and -135° . The T loop is located within

Table 5

Mean Indices of the Basic Magnitudes of a Normal Vectorcardiogram
According to M. I. Reiderman (1961)

Indices	Leads				
	I	II	III	IV	V
Maximum vector of the QRS loop (mv)	1.56 \pm 0.07	1.56 \pm 0.08	1.50 \pm 0.07	0.89 \pm 0.06	0.70 \pm 0.04
Maximum vector of the T loop (mv)	0.46 \pm 0.03	0.47 \pm 0.03	0.48 \pm 0.03	0.45 \pm 0.03	0.30 \pm 0.02
Arrangement of the QRS loop	+95.5° \pm 2.8°	+81.6° \pm 2.8	+93° \pm 1.8°	-119° \pm 11°	-92° \pm 7.7°
Location of the T loop	+103.4° \pm 3.3°	+90° \pm 3.1°	+64.4° \pm 4.0°	-121° \pm 8.8°	-119.0° \pm 10.6°
Angle QRS-T	15.4° \pm 2.8°	19.1° \pm 3.5°	26.5° \pm 3.2°	19.5° \pm 0.5°	43° \pm 7.6°
Area of the QRS loop (cm ²)	0.46 \pm 0.08	0.67 \pm 0.08	0.70 \pm 0.03	0.35 \pm 0.06	0.23 \pm 0.03
Area of the rectangle* (cm ²)	0.80 \pm 0.06	0.91 \pm 0.11	1.00 \pm 0.08	0.50 \pm 0.05	0.35 \pm 0.04

*According to the data of the author, the area of the rectangle recorded around the QRS loop can directly determine the area of the QRS loop: a measurement of the area of the rectangle is not difficult, technically speaking, while the determination of the area of the loop is bound up with a planimetric measurement.

the QRS loop and is sometimes outside of its limits; the angle of deviation between the maximum vectors of these loops does not exceed 40-50°.

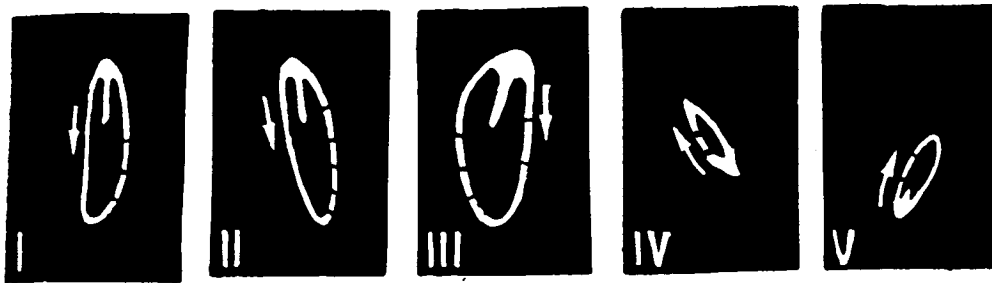


Fig. 70. A normal vectorcardiogram in five leads according to the I. T. Akulinichev system (explanation in the text).

As is evident from the description of a normal picture, the movement of the QRS loop changes with various revolutions of the heart about the anterior-posterior axis. It should also be noted that the shape of the loop also changes according to the rotation about the longitudinal axis: in the case of a clockwise rotation the loop becomes considerably more narrow in lead III and in the case of a counterclockwise rotation this narrowing is observed in lead II.

The above description of a normal vectorcardiogram in a precordial system is based on the data of a number of authors (Z. Z. Dorofeyeva and I. F. Ignat'yeva, 1958; M. I. Kechker and Sh. I. Shurgaya, 1958; E. A. Kyandzhuntseva and V. I. Makolkin, 1958). Table 5 gives the normal quantitative indices of M. I. Reiderman (1961); the standards which we have worked out are to be found on page 362.

2. A Normal Vectorcardiogram in Various Planes of the Cube System According to Grishman and Scherlis (Fig. 71)

On the horizontal plane the QRS loop, at the beginning, deviates a bit anteriorly and to the right; then its principal part moves to the left and a bit posteriorly counterclockwise. The maximum vector is located within the area of +30° to -30° and is equal to 3.2-19.4 mv. The maximum width of the loop is 1.0 to 12.4 mv. The T loop is within the QRS loop and its tracing is counterclockwise; the angle of deviation between vectors of loops QRS and T equals 11-49°. /193

On the sagittal plane the QRS loop deviates in the beginning somewhat anteriorly and often upwards and its principal part moves downwards and somewhat posteriorly in a clockwise manner. The maximum vector is located primarily in the area between +90° and +120° and equals 1.4-11.9 mv. The maximum width of the loop is 1.0-8.1 mv. The T loop is located within the QRS loop and its tracing is clockwise; the angle of deviation between the

maximum vectors of loops QRS and T is $63-86^{\circ}$.

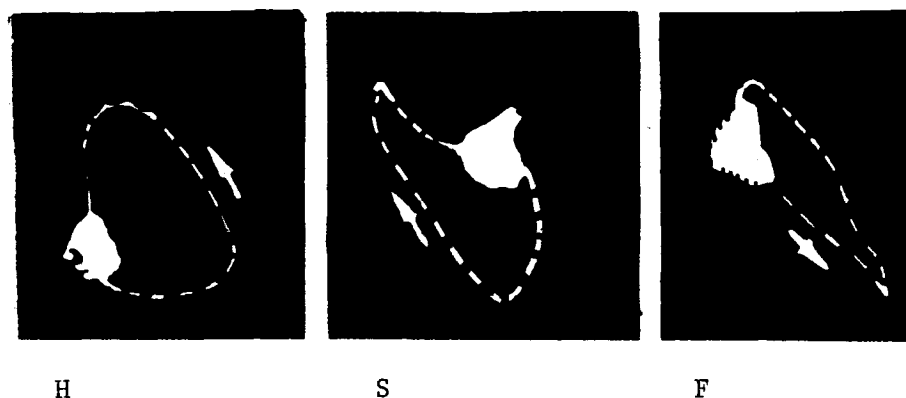


Fig. 71. A normal vectorcardiogram in three planes of the cubic system of Grishman and Scherlis (explanation in the text). Symbols: H = horizontal vectorcardiogram, S = sagittal vectorcardiogram, and F = frontal vectorcardiogram.

On the frontal plane the QRS loop can show a small initial deviation to the right and upwards, but its principal part moves downwards and to the left, clockwise or counterclockwise. Sodi-Pallares et al. (1957) found that the character of the direction of movement of the tracing of the ray in the frontal plane depends upon the rotation of the heart about its longitudinal axis. Grishman and Scherlis (1952) believe that usually a movement is observed clockwise in the case of the QRS axis from $+35^{\circ}$ to $+75^{\circ}$ and counterclockwise at axis QRS from $+35^{\circ}$ to 0° . However, Kornbluth and Allenstein, (1957), do not find any dependence between the direction of the tracing of the QRS loop and the deviation of the electric axis or the electrocardiographically-represented position of the heart (except in the case of a patient who is over 80 years of age). In a given plane the QRS loop can sometimes have a figure-eight shape. The maximum vector of the loop is 13.6-20.0 mv., its maximum width is 5.2-21.4 mv. The T loop is located within the QRS loop, its tracing is more often counterclockwise; the angle of deviation between the maximum vectors of the QRS and T loops is $15-39^{\circ}$.

Thus, the picture of the loops which is recorded by the two systems shows/194 that the spatial vectorcardiogram is as follows: the QRS loop is oriented to the left, downwards and somewhat posteriorly; the T loop is located anteriorly, downwards and to the right of the QRS loop. These loops are located approximately on one plane. The basic characteristic of the spatial arrangement of the loops does not undergo any significant changes under various physiologic conditions. Simonson, Nakagawa and Schmitt (1957) found some changes in the QRS and T loops in connection with various phases of the respiratory cycle, but they do not attribute any special significance to these changes. Schaffer considers that at various positions of the heart

the changes in the vectorcardiogram are caused, not only by the rotations of the heart about its axis, but also by changes in the characteristic of the organism, such as a volume conductor. The age factor has a definite significance. V. A. Astvatsatryan, (1960), noted that in healthy children from 7 to 14 years of age the resultant vector of the heart is directed from the top downwards and from the right to the left and anteriorly. Kornbluth and Allenstein (1957) found with age, a tendency towards orientation of the vector from the front-lower octant to the rear-upper octant. Burch, Golden and Cronvich (1958) spoke of a deterioration of the QRS loop in proportion to an increase in age and links this with a disruption in the depolarization process. O. V. Aleksandrov (1960) noted that the QRS loop decreases with age and assumes an ellipsoid form.

VI. CORRELATION BETWEEN THE ELECTROCARDIOGRAM AND THE VECTORCARDIOGRAM

There is no principal difference between the electrocardiographic method and the vectorcardiographic method. Both of them study the electromotive forces of the cardiac mechanism, the only difference being that the electrocardiographic method studies the electromotive force from the point of view of scalar magnitudes, whereas the vectorcardiographic method studies it from the position of vectorial principles. From this point of view there isn't anything in a vectorcardiogram which would not also be in a common electrocardiogram (Burch, 1958). Actually, as we have already seen, a vectorcardiogram gives summary information concerning the data of the two regular electrocardiographic leads (Mann constructed his monocardigram on this principle). These problems have been dealt with in the works of many authors (Duchosal, Groscurin and Sulzer, 1948; Goldberger, 1954; A. V. Holtzman, 1949, 1958; and others). Goldberger (1954) based all of his vectorcardiographic data on the principle of obtaining a vectorcardiogram from an electrocardiogram with the aid of a system of rectangular-coordinates. "Emslie-Smith" (1958) studied the horizontal and frontal vectorcardiograms with the aid of leads aVR, aVL, V_1 and V_4 . The problems concerned with ob-

taining a vectorial representation from ordinary electrocardiographic leads /195 are the subject of the study of vectorelectrocardiography to which the following chapter is devoted.

From the theoretical point of view, and to some extent also from the practical point of view, the obtaining of various leads of an electrocardiogram for use in a vectorcardiogram is of great interest. For this purpose we can use either the precordial system of recording or the cubic system of recording, but it is preferable to base oneself on the data of the cubic system since in this case the vectorcardiogram is recorded in definite planes which coincide with the planes of the axes of the electrocardiographic segments.

With respect to this problem we use the methodology of Grishman and Scherlis (1952). In order to derive from the vectorcardiogram an electrocardiogram in various leads we must keep in mind the following rules:

1. All of the planes of the vectorcardiogram and the segments of the electrocardiogram indicate from various positions the same phenomenon - i.e. the electric field of the heart. It can be considered that the horizontal vectorcardiogram represents the sum curve obtained from the data of the precordial leads, the sagittal vectorcardiogram is the sum curve of electrocardiograms in esophageal leads, and the frontal vectorcardiogram the sum picture of electrocardiograms in standard and/or amplified unipolar limb leads.

2. In order to determine any bipolar lead, the center of the loop (E) is connected to the center of the axis of that lead which, thus, divides up into two equal segments - a positive segment and a negative segment, depending upon the polarity of the lead. Thus, if we take leads I and II (Fig. 72a) it can be noted that that portion of the QRS loop which is projected onto the positive segment of the axis of the lead corresponds on the electrocardiogram to a positive fluctuation and that portion which is projected onto the negative segment of the axis of the lead expresses a negative fluctuation. In this case it should be remembered that the beginning and end of any electrocardiographic fluctuation is determined by the level of the zero point of the loop.

3. In order to determine any unipolar lead, the loop is placed in such a way that its zero point coincides with the electric center of the heart in the case of a precordial lead or with the central electrode in the case of a unipolar limb lead. Then the axis of the given lead is drawn and through the point of the electric center of the heart or of the central electrode, a perpendicular is drawn to this axis (Fig. 72b). If the loop moves to the side where the electrode is located then the fluctuation is recorded on the electrocardiogram as upwards, if the loop moves away from the electrode, then a downward fluctuation is obtained. In this case it is necessary to bear in mind that the beginning and end of any electrocardiographic fluctuation is determined by the level of the perpendicular line which has been drawn. /196

4. In order to determine the sequence of the waves on the electrocardiogram the projection of the loop must be begun from its initial side and must follow the tracing of the ray.

5. The amplitude of the waves of the electrocardiogram depends upon the extent of the projection of the loop on the axis of the given lead. The amplitude will be maximal if the maximum vector of the loop has a parallel direction towards the axis of the given lead and will decrease gradually as the maximum vector becomes perpendicular to the axis of the lead.

An electrocardiogram can be constructed on the basis of these rules.

The axes of the precordial leads are located at the level of the horizontal plane and, thus, these leads can be determined from the horizontal vectorcardiogram. On the cross section of the chest, passing through the electric center of the heart, the axis of the required precordial lead is drawn (for this purpose the proper chest position is connected by a straight

line to the electric center of the heart). Then, placing the horizontal vectorcardiogram in such a way that its zero point coincides with the electric center of the heart, it is possible to determine, in accordance with the rules which have been given, a picture of an electrocardiogram with the given precordial lead (Fig. 73a).

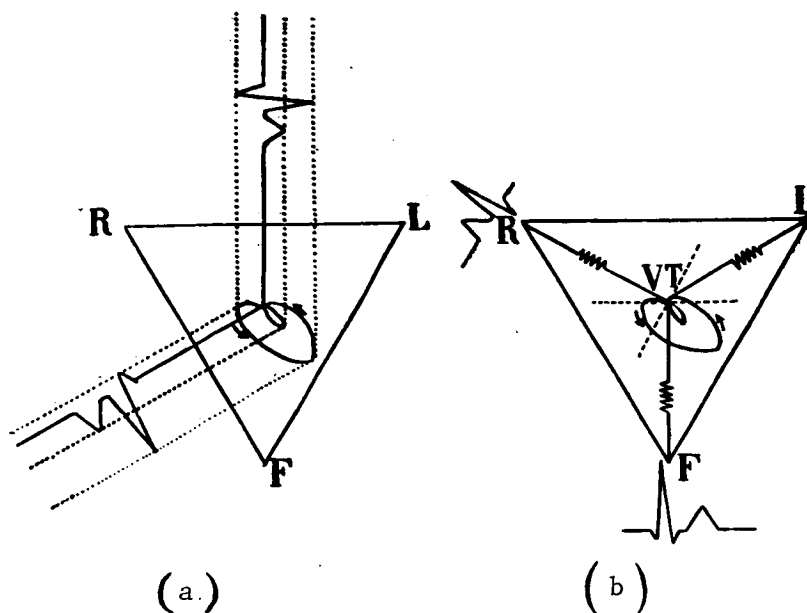


Fig. 72. Correlation between an electrocardiogram and a vectorcardiogram: (a) determination of a bipolar lead from the vectorcardiogram; (b) determination of a unipolar lead from the vectorcardiogram.

The axes of the esophageal unipolar leads coincide with the sagittal plane and the axis of the lowest esophageal lead corresponds to the axis of lead VF. The sagittal vectorcardiogram is placed in the point of intersection of the axes of the esophageal leads and, according to the rules which have been given, the picture of the electrocardiogram in the corresponding esophageal unipolar lead (Fig. 73b) is determined.

The axes of the standard leads and the unipolar limb leads are located at the level of the frontal plane and for this reason these leads can be determined from a frontal vectorcardiogram. With this purpose in mind, the vectorcardiographic loop is placed in the center of a hexaxial system and, according to the rules which have been given, the picture of an electrocardiogram with the corresponding limb leads (Fig. 73c) is determined.

It should be kept in mind that an electrocardiogram which is determined from vectorcardiographic loops is somewhat conditional and cannot be matched completely with a true picture in a given lead. For instance, how is it possible to obtain an accurate precordial curve from the horizontal loop if all of the chest positions are not arranged on the horizontal plane of the electric center of the heart? The same may also be said with respect to the

other leads, since in man, the esophageal leads cannot be situated on one plane and the frontal plane of a cubic system does not coincide with the frontal plane of the Einthoven triangle. In addition to this, pictures of the P wave, segments P-Q and RS-T are not obtained on these curves.

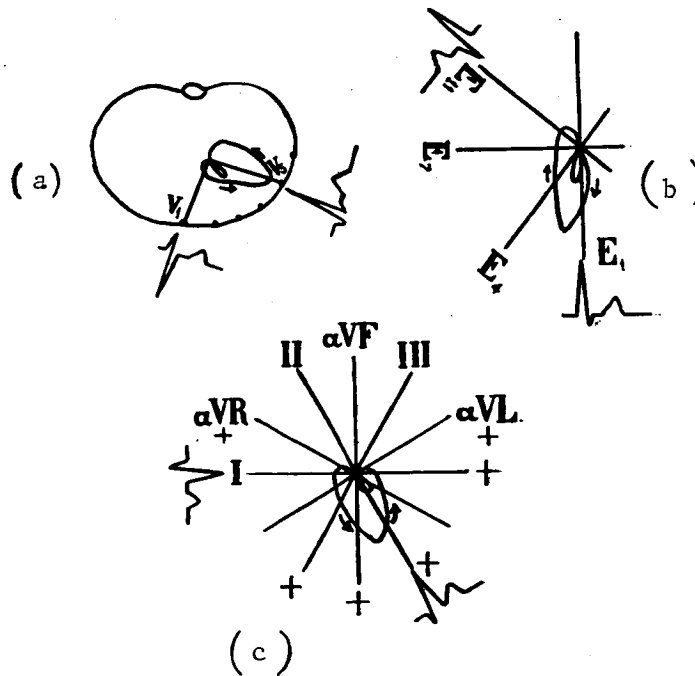


Fig. 73. The correlation between an electrocardiogram and a vectorcardiogram: (a) obtaining of an electrocardiogram in precordial leads from a horizontal vectorcardiogram; (b) obtaining of an electrocardiogram in esophageal unipolar leads from a sagittal vectorcardiogram; (c) obtaining of an electrocardiogram in limb leads from a frontal vectorcardiogram (explanation in the text).

VII. PATHOLOGICAL CHANGES IN A VECTORCARDIOGRAM

In the case of pathologic conditions of the heart, various changes may be observed on the vectorcardiogram. These changes may be grouped schematically in the following manner (we are primarily concerned with the QRS loop).

1. Changes in the shape of the loop when it assumes the shape of a figure-eight, a sphere, a pear, a triangle; when a positive pole appears; when indeterminate and odd figures are drawn, and so on.
2. Changes in the principal contours of the loop when there appears on the tracing a depression, coil-shape, tapering, or when there appears a posi-

tive loop-formation, and so on. Such changes are often observed in focal changes, and M. N. Tumanovskiy (1958) and I. I. Bykov (1958) assign an electrophysiologic significance to them.

3. Changes in the position of the loop when the location of its maximum vector is changed to the point of displacing it into the opposite quadrant of the coordinate system.

4. Changes on the side of increase or decrease of the maximum vector or the width of the loop.

5. Increase or decrease in the area of the loop, since this may sometimes occur in such a way that the maximum vector of the loop does not undergo any particular changes.

6. Changes in the tracing of the movement of the loop.

7. Changes in the angle of deviation between the maximum vectors of the QRS and T loops since the increase in this angle can be to such a degree that the T loop appears outside of the limits of the QRS loop. It can be stated that the increase in this angle often precedes the occurrence of pathologic changes in the T wave.

8. The QRS loop may become open. It is evident that in a normal loop there is one general zero point for the QRS and T loops. A picture of this type is caused by the fact that an electromotive force does not arise in the tension phase during systole of the ventricles. Under several pathologic conditions (in particular in the case of hypoxia and myocardial infarction) an electric current develops in this phase and an RS-T vector appears as a result of this; in the case of this loop, QRS remains open because the T loop does not start from the zero point. The amplitude and the spatial arrangement of the RS-T vector determine the character and extent of displacement of the RS-T segment from the isoelectric lines on the electrocardiogram. /199

VIII. VECTORCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE SYNTHETIC ELECTROCARDIOLOGIC COMPLEX

1. Hypertrophy

A. Auricular hypertrophy. In the case of left auricular hypertrophy the P loop is wide and open, and the maximum vector is directed posteriorly, inferiorly and to the left. In the case of right auricular hypertrophy a rather wide and open P loop is observed having a maximum vector which is oriented anteriorly and inferiorly. The angle of deviation between the Ta and P vectors is 180° . These data have been obtained by Sano, Hellerstein and Vaid (1957) by using the differential vectorcardiographic method.

B. Ventricular hypertrophy. In the case of left ventricular hypertrophy the left ventricular vectors prevail over the right ventricular vectors and, as a result, a displacement of the QRS loop to the left and posteriorly is

observed on the side of the hypertrophied ventricle. In the majority of cases an increase in the maximum vector and in the area of the QRS loop resulting in an increase in the total electromotive force of the heart is obtained, but the normal configuration of the loop remains without significant changes. An opening of the QRS loop is observed. The T loop increases and its maximum vector moves to the opposite quadrant. These changes occur gradually. M. B. Tartakovskiy (1960) differentiates four periods.

During the first period, the characteristic increase in the maximum vector and in particular in the area of the QRS loop is observed. A great deal of importance is attached to this moment by a majority of authors (M. I. Kechker, 1958; Zh. A. Teslenko, 1961, and others). Moreover, in the final portion of the loop there is observed a small swelling which gradually increases, in the form of an additional pole, and moves further away from the zero point of the loop.

During the second period, an opening in the QRS loop occurs.

In the third period, already characterized by an altered background, the T loop is displaced to the opposite quadrant as regards the direction of the QRS loop. A discordance of this kind is a sign of marked hypertrophy; it is not observed in the case of slight degree of hypertrophy.

The fourth period is when the characteristic signs of hypertrophy are not /200 present, since diffuse dystrophic changes taking place in the myocardium cause a change in the vectorcardiographic pattern.

In persons afflicted with hypertrophy we observed practically identical pictures of left ventricular hypertrophy. In the vectorcardiogram (Fig. 74) there are often observed changes in the form and arrangement of the QRS loop, an increase in the QRS-T angle, and arrangement of the T loop beyond the limits of the QRS loop to the point of appearance of complete discordance among them. The tracing of the QRS loop quite often does not change. These changes increase gradually as the severity of the disease develops. A change of this type is easily determined from the principal indicators of the QRS loop. This change gradually moves to the left; a gradual increase in the maximum vector, maximum width and area of the loop is observed (Table 6). Here the spatial arrangement of the loop moves to the left, inferiorly and posteriorly; another arrangement is seldom encountered. We observed a deformation of the loops in the presence of a bundle branch block (His), marked cardiosclerosis and focal changes in the myocardium. M. I. Reiderman (1961) also points this out.

In persons suffering from hypertrophy, a widening of the QRS loop is often observed with a mirror image in leads I and III and a sharp narrowing of the QRS loop in lead II. This pattern can be explained by the rotation of the heart about the longitudinal axis counterclockwise as a result of left ventricular hypertrophy. E. A. Kyandzhuntseva and V. I. Makolkin (1960) also /201 wrote about this. In addition, in the initial stages of the development of hypertrophy, a widening of the loop in the area of the apex is often observed.

I. F. Ignat'yeva (1959) indicated the occurrence of an additional pole and a gradual increase in it on the side of the opposite quadrant. A greater development of this pole in lead IV is linked by the author to the recording of predominant apex forces in that lead (the area of the apex becomes hypertrophied earlier than the other portions of the heart). It is evident from the data given that the early stages of the development of left ventricular hypertrophy are easily determined in leads I, III and IV.

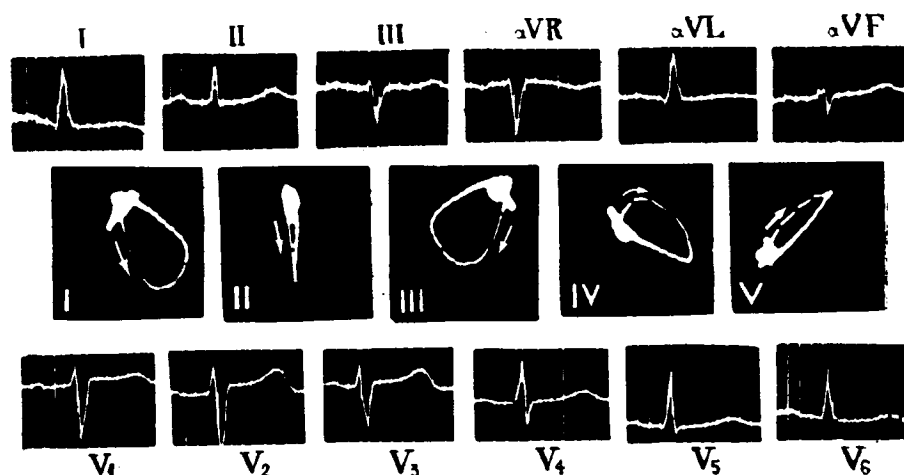


Fig. 74. Left ventricular hypertrophy. A vectorcardiogram and electrocardiogram of a patient, A. G., 64 years of age, with the following diagnosis: hypertensive disease in stage 1b, arteriosclerotic cardiosclerosis. The left ventricular hypertrophy appears in a stage which is not acute.

Table 6

Some Indicators of the QRS Loop in Lead I During Various Stages of Hypertensive Disease

Component of the QRS Loop	Stages of the Disease		
	Primary	Secondary	Tertiary
Sector of arrangement (in degrees) in the system of coordinates	+39.81±11.66	+33.52±12.33	+31.1±11.04
Maximum vector (in cm.)	1.95±0.79	2.13±0.87	2.63±1.04
Maximum width (in mm.)	5.13±3.76	7.59±3.98	9.20±5.01
Area (in cm ²)	0.78±0.63	1.37±1.06	2.44±1.48

V. I. Makolkin (1960) noted in the case of left ventricular hypertrophy two types of vectorcardiographic changes. The first type, which is more often encountered in persons with less marked changes in the myocardium and with mitral defects, is characterized by normal orientation of the QRS loop but with marked end deviation to the left and posteriorly. The second type is encountered more often in persons having more marked changes in the myocardium and with aortic defects and is characterized by a lengthening of the QRS loop with considerable deviation to the left and posteriorly and the development of crosses.

According to the data of Grishman and Scherlis (1952), in the cubic system of recording on the horizontal plane, the QRS loop, after a slight deviation anteriorly and somewhat to the right, moves counterclockwise to the left and posteriorly. The maximum vector is located in the sector from -30° to -50° . On the sagittal vectorcardiogram the QRS loop is directed posteriorly, but has an initial deviation anteriorly and inferiorly. The maximum vector is located in the sector from $+120^{\circ}$ to $+170^{\circ}$. The direction of the tracing of the loop is clockwise, although Sodi-Pallares (1956) observes /202 more often a counterclockwise movement of the ray. On the frontal plane the loop moves clockwise or counterclockwise to the left, and its maximum vector is located in the sector from $+5^{\circ}$ to $+40^{\circ}$. In this plane Bilger (1957) distinguishes two types of QRS loops - a narrow QRS loop and a wide QRS loop directed to the left and upwards. In both types the QRS and T loops are arranged discordantly.

In right ventricular hypertrophy the right ventricular vectors prevail, as a result of which there is observed a displacement of the loop to the right and anteriorly on the side of the hypertrophied ventricle. I. T. Akulinichev (1960) noted that the earliest sign of right ventricular hypertrophy is a decrease in the angle of deviation of the QRS and T loops. Further on, as a result of the rotation of the heart clockwise about the longitudinal axis, there is observed a widening of the QRS loop in leads I and II and a narrowing of the QRS loop in lead III; to this is added an opening of the QRS loop and a widening of the T loop.

The vectorcardiographic characteristic of right ventricular hypertrophy (Fig. 75) has been well studied by M. I. Kechker (1960) who distinguishes the following three kinds of curves:

1. In marked hypertrophy the QRS loop is oriented to the right and posteriorly, the area of the loop is increased, and often there is a discordant /203 arrangement of the T loop. The tracing of the ray in lead I is clockwise and it is counterclockwise in lead III.

2. The principal portion of the QRS loop does not increase, there is an increase in its final portion which is oriented to the right, upwards and anteriorly, less often posteriorly, as a result of a deceleration of the conduction. The tracing of the ray in lead I is clockwise and it is counterclockwise in lead III.

3. The initial deviation of the QRS loop, as a result of summation of

the potentials of the right ventricle and the intraventricular conductance, increases considerably; it is oriented anteriorly and to the right. The tracing of the loop is counterclockwise in lead I and clockwise in lead III.

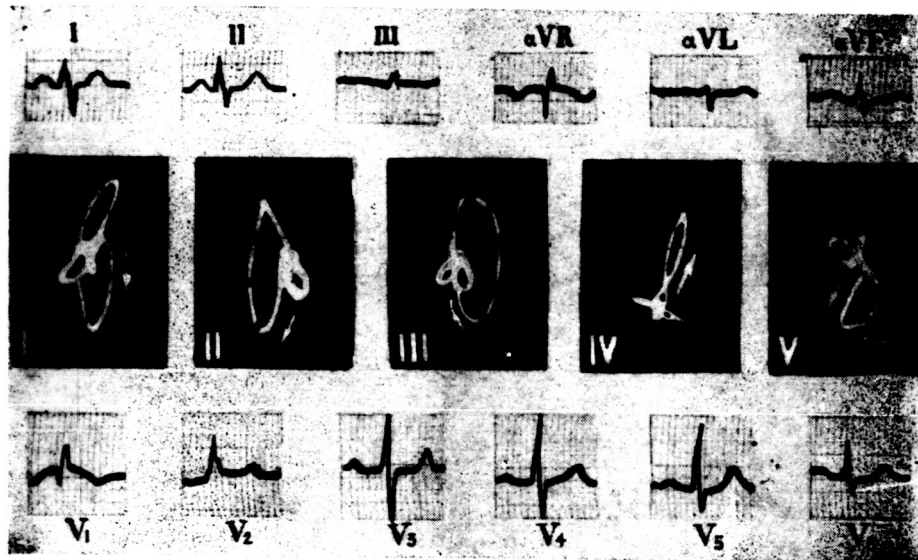


Fig. 75. Right ventricular hypertrophy. A vectorcardiogram and an electrocardiogram of a patient, E. P., 23 years of age, with the following diagnosis: mitral defect with predominance of stenosis (explanation in the text).

Based on the data of a number of authors, Grishman and Scherlis (1952) also distinguished three types of vectorcardiograms according to the cubic system.

1. In the case of slight or moderate right ventricular hypertrophy, on the horizontal plane of the initial portion, the QRS loop is oriented to the right and anteriorly; then the loop deviates to the left and posteriorly, and the final portion of the loop moves clockwise to the right and anteriorly. On the sagittal vectorcardiogram the QRS loop is oriented counterclockwise anteriorly and its final portion is oriented somewhat superiorly. The loop is narrow and sometimes has a figure-eight shape. On the frontal plane the QRS loop shows an initial upward deviation and its final portion is oriented anteriorly and somewhat posteriorly. The tracing of the ray is clockwise.

2. In the case of slight hypertrophy, on the horizontal plane there is noted an initial deviation of the QRS loop to the right and anteriorly, and then somewhat to the left, and further on, the deviation is clockwise to the right and anteriorly. Such a movement of the loop indicates a predominance of right ventricular potentials. On the sagittal vectorcardiogram the QRS loop is oriented counterclockwise anteriorly, inferiorly or upward. On the frontal plane the QRS loop moves to the right, clockwise.

3. In marked right ventricular hypertrophy, on the horizontal plane a small initial deviation to the right is noted; then to the left. The principal part of the loop moves clockwise to the right and somewhat posteriorly and sometimes has a figure-eight shape. On the sagittal vectorcardiogram the loop is narrow, sometimes in the shape of a figure-eight. It moves clockwise upward and anteriorly or posteriorly. On the frontal plane the QRS loop is oriented upward, then it may be oriented clockwise to the right or counterclockwise to the left.

Bilger (1957) also distinguishes such types but he also distinguishes a fourth type in which case the QRS loop is narrow. It is oriented on the horizontal plane to the right and an anterior deviation is scarcely noted. On the frontal plane there is a strong lengthening of the loop to the right and upward.

Thus, it can be concluded that in proportion to the development of right ventricular hypertrophy, a gradually increasing deviation of the QRS loop to the right, anteriorly and upward occurs. In acute stages of hypertrophy an orientation of the loop to the right posteriorly or to the left and upward is obtained. It should be noted that as a result of these dynamics, the T loop gradually assumes a discordant position. /204

In combined ventricular hypertrophy vectorcardiography can be of definite assistance since the described vectorcardiographic criteria of hypertrophy of the individual ventricles can be expressed simultaneously. Thus, according to the data of V. I. Makolkin (1960), under these conditions there is observed a marked initial deviation of the QRS loop anteriorly and to the right and a final deviation posteriorly and to the left. The tracing of the rotation of the loop does not change. Dack, Beregovich and Bleifer, (1960), consider that in determining combined hypertrophy, vectorcardiography has a great advantage over electrocardiography.

2. Coronary Insufficiency

A. Acute coronary insufficiency without infarction (with the symptoms of stenocardia). The hypoxic phenomena developing in the myocardium primarily have an effect on the tension and repolarization phase of the electrical activity of the ventricles. On the vectorcardiogram various changes are detected in the shapes of the QRS and T loops. The QRS loop remains open, the angle of deviation between loops QRS and T changes, the QRS loop loses its flat contour and various disruptions occur, particularly those of tapering, which are, according to the data of M. I. Tumanovskiy (1958), temporary signs of focal infection of the myocardium. The author also points to a decrease outside of the beginning of the T loop and marked asynchronism of the loops. I. I. Bykov (1959) distinguishes three kinds of vectorcardiograms which correspond to the angioneurotic, arteriosclerotic and transitional forms of stenocardia. In all of these kinds of vectorcardiograms there are expressed, to one extent or another, notchings, taperings, coil-shapes or other changes in the contours of the QRS loop. V. I. Klemenov (1961) observed the same changes with considerable decrease in the QRS and T loops in the case of arteriosclerosis of the coronary arteries.

B. Acute coronary insufficiency with myocardial infarction. In this case the vectorcardiographic changes are particularly interesting. The mechanism of occurrence of these changes is reduced basically to the following. It is evident that the vectorcardiographic loop is a reflection of the conventional electric field of the heart which has some form and relative stability thanks to the creation of certain correlations between the numerous vectors of the heart. When infarction develops, this vector balance is disrupted since the /205 necrotic portion becomes a dead zone and does not take part in the creation of the electric field of the heart. As a result of these changes, the electric forces which are opposite the necrotic portions of the heart appear in an unbalanced state and a displacement of the loop on the side opposite the necrotic part is obtained. This displacement can be shown by means of a so-called infarcted vector which is situated further from the necrotic portion towards the direction of the portions opposite to it (Fig. 76). Thus, a myocardial infarction appears on the vectorcardiogram as a corresponding change in the loop. For instance in the case of anterior infarction the QRS loop will be displaced posteriorly. In the case of posterior infarction it is displaced anteriorly. In the case of combined infarction it is displaced in the direction of the sum infarcted vector. It should be kept in mind that this characteristic displacement of the loop will not be detected only in that plane with respect to which the infarcted vector has a perpendicular direction. Besides this, the character of the final curve will still also depend upon the condition of the myocardium prior to the occurrence of the infarction, the development of the perifocal block around the necrotic node, and other factors.

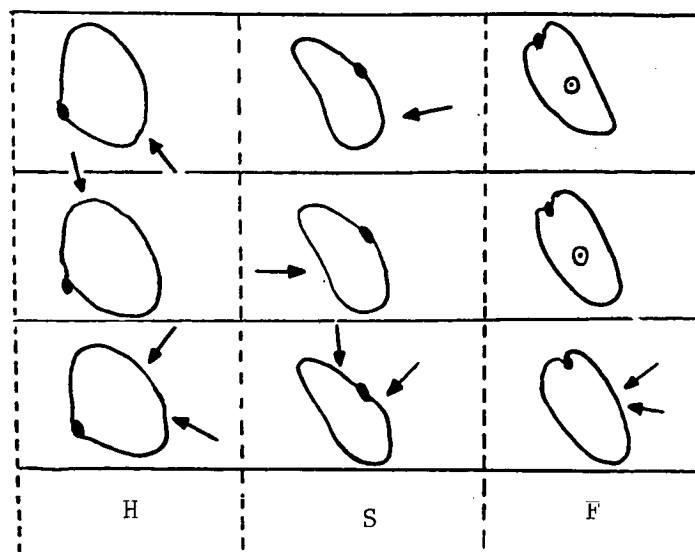


Fig. 76. Direction of action of the infarcted vector on the horizontal (H), sagittal (S) and frontal (F) vectorcardiogram of Grishman and Scherlis. The first series from the top is anterior infarction, the second series is posterior infarction, and the third series is anterolateral infarction. The perpendicular direction of the vector is indicated by a circle with a point in the center.

In myocardial infarction a sharp deformation of the QRS loop sets in, a /206 distortion takes place, and additional loop formation and other changes occur (V. S. Gasilin and Yu. P. Mironov, 1960). In addition to this, the spatial arrangement of the loops changes. The QRS loop remains open and a deformation is observed in the T loop. A characteristic feature is the pattern of the initial portion of the QRS loop changes. In the descriptive explanation of Donzelo, Milovanovich and Kaufmann (1950), an initial "allodromy" is created, i.e. a disruption of the excitation process of a kind which should not be explained by the spatial arrangement of the heart. We must agree with Lem (1957) that the classical pattern of myocardial infarction is characterized by the following features: the initial 0.04 QRS vector is directed further from the infarcted node, the RS-T vector is directed to this portion and the T vector is directed beyond it.

Based on the data of E. A. Kyandzhunitseva and V. I. Makolkin (1958), Grishman and Scherlis (1952), we give a description of the vectorcardiograms for the locations of infarctions which are most often encountered. These data refer to the relatively late period of development of infarction since no one has as yet been able to record a vectorcardiogram during the first hours after the onset of an acute attack.

a. Anterior infarction (Fig. 77). The QRS loop is directed posteriorly and its initial portion is shifted upward to the right and posteriorly.

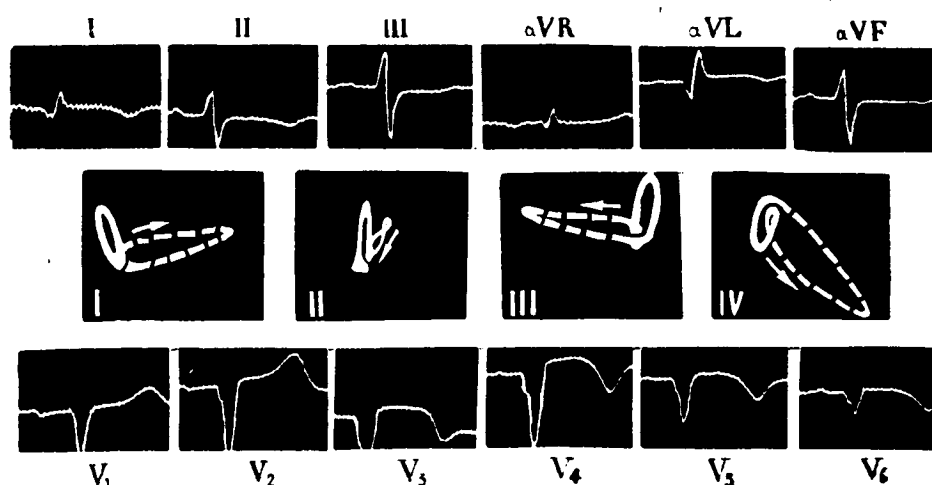


Fig. 77. Anterior infarction. A vectorcardiogram and an electrocardiogram of a patient, S. T., 52 years of age, with the following diagnosis: anterolateral infarction, hypertensive disease of stage 1b (explanation in the text).

The initial portion of the QRS loop in lead I of the precordial system is displaced superiorly to the right. Occasionally loop QRS is narrow or has a figure-eight shape. It is located in the sector from $+20^{\circ}$ to -40° . Loop T is usually displaced upwards and to the right. In lead II a deviation of the

loop to the right is observed. The deviation from the frontal surface is particularly distinct in the initial portion of the QRS loop and in its downward curvature. The QRS loop is situated in the sector from $+90^{\circ}$ to $+200^{\circ}$. The rotation of the loop in the initial portion goes clockwise and the loop has a figure-eight shape. In the case of a change in the larger portion of the loop, the rotation goes counterclockwise. Loop T is larger and moves in the direction of the initial portion of loop QRS. In lead III the loop deviates to the left, particularly in its initial portion. The loop is situated in the sector from $+125^{\circ}$ to $+170^{\circ}$ and may have the base of a figure-eight. In the case of a change in the larger portion of the loop, the rotation of not only the initial portion, but also of the entire loop is counterclockwise. The changes in the T loop are the same as in lead II. In leads IV and V no additional indications whatsoever are observed. Here there is observed a marked displacement of the QRS loop to the left and posteriorly. In the case of anterolateral infarction the deviation of the QRS loop to the right predominates, and in the case of an anteroseptal infarction the deviation of the QRS loop posteriorly predominates. /207

In the case of the cubic system of recording, on the frontal plane of the vectorcardiogram no changes occur, since the infarcted vector is oriented perpendicularly to this plane. On the horizontal plane the QRS loop is directed first of all to the right and anteriorly and then deviates sharply posteriorly and forms a figure-eight clockwise or counterclockwise. On the sagittal plane the QRS loop is directed anteriorly at the beginning and then posteriorly clockwise. In anteroseptal infarction the loop rotates counterclockwise in this plane and in anterolateral infarction the sagittal vectorcardiogram may remain unchanged (Sodi-Pallares, 1956).

b. Posterior infarction (Fig. 78). The QRS loop is deviated anteriorly. The vectorcardiographic differentiation of the anterodiaphragmatic infarction and anterolateral infarction is of special interest.

In the case of anterodiaphragmatic infarction, the QRS loop, especially its initial portion, deviates somewhat anteriorly and upward. According to the data of I. T. Akulinichev (1960), the initial portion of the loop in leads I, II, III and V is displaced anteriorly, upward and to the right, and its basic portion in leads I, IV and V to the left, in lead II anteriorly, and in lead III in a narrow pole posteriorly. The direction of the tracing of the loop seldom changes and the loop assumes a figure-eight shape. The T loop increases and is directed upwards and somewhat to the left.

In the case of anterolateral infarction, the QRS loop deviates anteriorly and to the right. For differentiating the anterolateral infarction from the anterodiaphragmatic infarction, E. A. Kyandzhuntseva and V. I. Makolkin (1958) very substantially attribute importance to plane III in which, in the case of the first position, the initial portion of the QRS and T loops is directed sharply to the right and upwards, and in the case of the second position sharply to the left and upwards. As a differential-diagnostic sign, I. S. Kun (1961) notes the fact that in lead III in anterodiaphragmatic infarction the initial portion of the QRS loop is situated to the left of the zero point and rotates counterclockwise, and in anterolateral infarction it /208

is situated to the right of the zero point and moves counterclockwise. In anterolateral infarction it becomes situated to the right of the zero point and rotates clockwise.

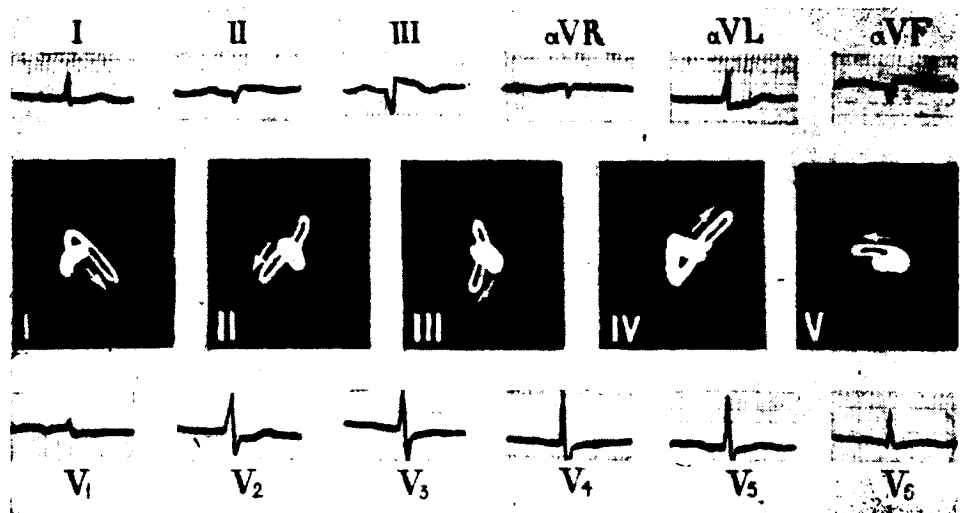


Fig. 78. Posterior infarction. A vectorcardiogram and an electrocardiogram of a patient, Kh. E., 63 years of age, with the following diagnosis; posterior infarction, atherosclerotic cardiosclerosis (explanation in the text).

In the cubic system of recording, the anterodiaphragmatic infarction does not appear on the horizontal plane since the infarction vector acts perpendicularly on this plane. On the sagittal vectorcardiogram the initial portion of the QRS loop is directed upwards and anteriorly counterclockwise. The remaining portion of the QRS loop may rotate clockwise, moving inferiorly and somewhat posteriorly. On the frontal vectorcardiogram the initial vectorial allodromy is usually displaced clockwise upwards.

In anterolateral infarction the QRS loop is more often displaced clockwise to the right and anteriorly on the horizontal plane. On the sagittal plane it moves clockwise or, according to Sodi-Pallares (1956), counterclockwise anteriorly, and on the frontal plane it moves counterclockwise to the right.

E. A. Kyandzhuntseva and V. I. Makolkin (1958) emphasize the significance of lead II in differentiating anterior infarctions from posterior infarctions. In anterior infarction the QRS loop in lead II deviates to the right, and the QRS loop or its initial portion rotates clockwise, loop T is displaced to the right upwards. In posterior infarction in this lead a deviation of the entire loop to the left and an orientation of its initial portion and of loop T to the left and upwards are observed. /209

According to the data of I. S. Kun (1961), the position of the necrotic node predominantly in the region of the lateral wall of the left ventricle is easily seen in leads I and III where there is noted a marked shifting of loops QRS and T to the right. If the node also spreads onto the portion of the anterior surface of the heart adjacent to the lateral wall, then a deviation of QRS posteriorly is observed, and in lead II the orientation of the loop is inverted.

In the cubic system of recording, diagnostic signs appear in the horizontal and in the frontal planes since the infarct vector has a perpendicular direction towards the sagittal plane. On a horizontal vectorcardiogram the QRS loop deviates to the right and somewhat anteriorly. Then the major portion of the loop moves counterclockwise posteriorly to the left or clockwise to the right. On the frontal plane the initial deviation moves to the right, and the remaining portion moves counterclockwise vertically inferiorly and to the left.

3. Bundle of His Branch Block

As a result of the disruption of the intraventricular conductance the time courses are situated close to one another on the vectorial curve, the tracing of the loop is thick, and the loop moves to the side of the diseased myocardium.

In a left bundle of His branch block (Fig. 79) the QRS loop loses its usual form, deformations in the loop occur and the QRS loop does not terminate at the zero point. The maximum vector of the QRS loop is directed posteriorly to the left and upwards, and loop T has a discordant position. In all of the precordial leads there is observed a narrowing and crossing of the QRS loop. On the horizontal vectorcardiogram of the cubic system the loop has a figure-eight shape, rotates clockwise and is directed posteriorly and to the left. On the sagittal plane the loop is directed posteriorly and upwards clockwise. On the frontal vectorcardiogram the QRS loop is displaced counterclockwise to the left and upwards. Sometimes the loop rotates clockwise, especially when it has a vertical position. In all of these cases a slowing down in the conductance is noted in the middle and in the initial portions of the loop, and, as was noted by Enescu et al. (1953), the swollen portion shifts between the two branches of the loop which have a normal rate of development. V. F. Sysoyev (1960) considers that the presence of sudden decelerations in the rate of movement of the ray at the beginning in the apex or in the end of the QRS loop should be given a differential-diagnostic significance in fixing the boundaries of the left branch block from the hypertrophy of the left ventricle. According to his opinion, the differential-diagnostic position which in the left bundle branch block of the loop moves clockwise in the horizontal plane and moves counterclockwise in left ventricular hypertrophy is questionable. Frimpter, Scherr and Ogden, (1958), turned their attention to the initial portion of the loop which is 0.06-0.28 mv. on the horizontal plane and which is directed to the left and anteriorly. It is 0.06-0.33 mv. on the sagittal plane and is directed anteriorly and inferiorly, and is 0.04-0.30 mv. on the frontal plane and develops poorly. Cabrera et al. (1958), indicate a notched value R in the center of the loop.

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In a right bundle of His branch block (Fig. 80) there is observed in all of the leads and in all of the planes a normal course of the tracing even though Cabrera et al. (1958), note that on the horizontal plane, beyond the dependency upon portion R, the S loop rotates counterclockwise. In this case, large changes in the shape of the loop are not observed, but on the final portion of the loop an additional pole which has an indefinite shape is observed and recorded relatively slowly, and is directed to the right and anteriorly. This final portion of the QRS loop is easily detected in the horizontal plane. A pattern of this kind can be explained by the fact that /211 during the course of the first few seconds there only occurs a depolarization of the healthy ventricle and the QRS loop is recorded as usual. However, since the depolarization of the right ventricle also begins a bit later, there also appears in the vectorial field those forces of the ventricle which bring about the recorded changes in the final portion of the loop. It may be roughly said that in the case of a right branch block the initial portion of the loop corresponds to the electric force of the left ventricle and the final portion of the loop is recorded basically on account of the activity of the right ventricle.

In practice, cases of myocardial infarction in conjunction with bundle of His branch blocks are encountered rather often. A diagnosis of combinations of this kind is difficult, particularly if the background on which this combination takes place has undergone a considerable change.

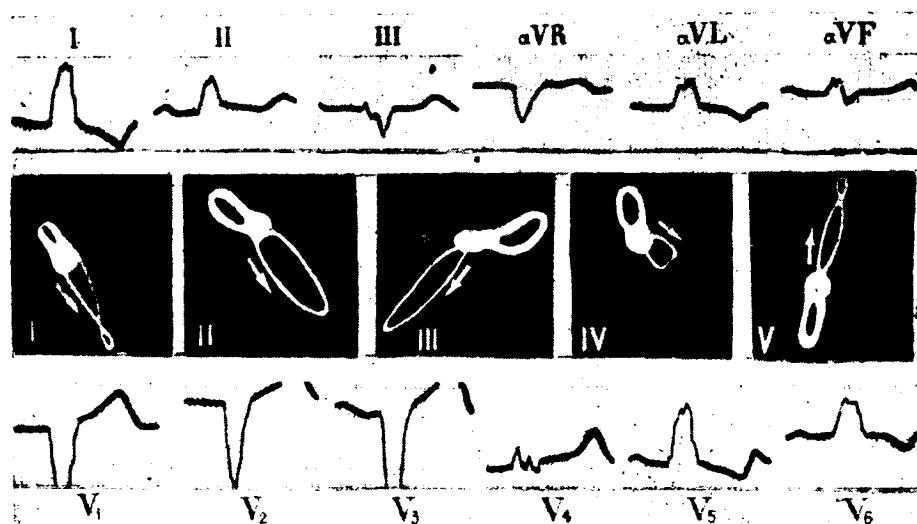


Fig. 79. Left bundle of His branch block. A vectorcardiogram and an electrocardiogram of a patient, V. A., 36 years old, with the following diagnosis: hypertensive disease of the stage Ib (explanation in the text).

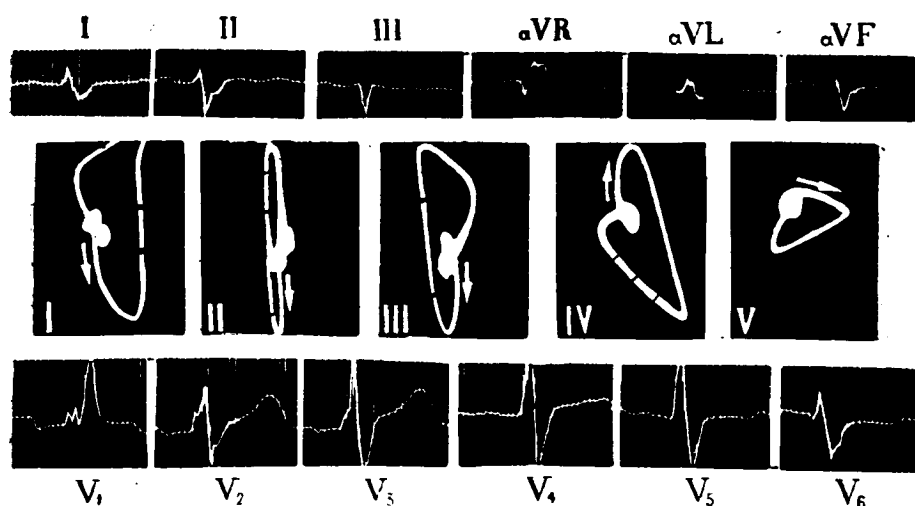


Fig. 80. Right bundle of His branch block. A vectorcardiogram and an electrocardiogram of a patient, A. R., 58 years old, with the following diagnosis: hypertensive disease of the stage IIIa; atherosclerotic cardiosclerosis (explanation in the text).

It is rather complicated to recognize a myocardial infarction which develops on the background of a left branch block. Only circumstantial evidence is of any assistance in this case. Thus, on the QRS loop initial changes which are characteristic of infarction are observed, and final changes which are related to the block (notches appearing in place of a relatively flat course in the case of the block) are seen. I. T. Akulinichev (1960) noted that the T loop decreases and, as a result of a still greater breaking of the QRS loop, it acquires the shape of a horseshoe. I. I. Bykov (1958, 1959) introduces certain criteria for diagnosing the left bundle of His branch block in the cases of various positions of the infarction, but even these criteria cannot give an accurate indication as to the presence of this pathological combination.

In the case of a development of myocardial infarction on the background of the right bundle of His branch block, the diagnosis is relatively simpler. In the beginning of the loop, a deviation which is typical for infarction is observed, and, in the end of the loop, there is an additional pole which is characteristic for this block. A recognition of this combination becomes difficult in the case of several positions of the necrotic focus. /212

IX. CONCLUSION

We have noted in a very short and concise manner the significance of this method of investigation only under a few circumstances: (hypertrophy, infarction, bundle branch block) so that, in our opinion, it is precisely here that this method can supplement electrocardiographic investigation and find its place in synthetic methodology. The role of vectorcardiography is not very great

with respect to the determination of anterior infarctions. This method is of definite assistance in the diagnosis of anterolateral infarctions and anterodiaphragmatic infarctions and also in cases of a combination of infarctions and branch blocks. Although Howitt and Lawrie (1960), speak of the fact that in the majority of cases of infarction the vectorcardiographic method is less valuable when compared with the electrocardiographic method. Karni (1957), on the other hand, notes that vectorcardiographic indicators present a better clinical picture of myocardial infarction. Vectorcardiographic investigation contributes considerably to the detection of the presence of right ventricular hypertrophy (Donoso et al. 1957); (Silverblatt et al. 1957), especially during its early stages (Dahl and Simonson, 1953); (M. I. Kechker, 1960; G. G. Didebulidze, 1960) and in combination with a right branch block (Hamer, 1958). Z. Z. Dorofeyeva (1959) attributes a great deal of importance to the vectorcardiographic method in the determination of diffused changes in the myocardium, in particular in cases of rheumatic carditis.

The multitude and the great variety of contemporary methods of recording a vectorcardiogram cannot contribute favorably to the further development and propagation of the vectorcardiographic method, nor to its broad popularization, and we agree with Burger, van Brummelen and van Gerpen (1961) that the future of vectorcardiography depends upon the extent of the conformity or non-conformity between these various systems. Duchosal (1958) looks at this problem in an optimistic manner and considers that a mutual understanding between the various authors leads to the elaboration of a sole system of recording in the field of vectorcardiography.

We cannot concur with either the opinion of those authors who overstate the value of vectorcardiography or of those authors who understate its value. Besides, we do not consider it proper to make a comparison of the significance and the value of the two methods (electrocardiography and vectorcardiography)/²¹³ since the latter method does not as yet have a long history and is actually in the developmental stage. We also cannot object to the vectorial principle of recording, as is done by Grant (1957), since we consider that the unipolar electrode does not record the local potential of the portions of the heart which correspond to it, but rather, the total pattern of the vectors of the heart which takes place in a given portion of the heart. We find that in attaining a solution to given problems, vectorcardiography, as a new division of electrocardiology, adds to the data obtained by the classical method of electrocardiology but cannot change the classical method. It suffices to say that in the case of detecting the various disruptions in the rhythm of the heart, vectorcardiography presents rather scanty data. The utilization of vectorial principles in the field of electrocardiology should in every way be welcomed since this marks a new way of introducing accurate physical and mathematical data into cardiology, enriches the approach and the thinking of specialists, and opens up, without a doubt, new horizons in electrocardiology.

SPATIAL VECTORCARDIOGRAPHY

I. INTRODUCTION

The point discussed in the previous chapter was that all the basic leads of an EKG could be derived from vectorcardiograms of various planes, and that the various EKG leads could be used to form a vectorcardiographic loop. There is still another method that can be used when an EKG is recorded with 12 leads. These EKG's, however, are not interpreted by the usually accepted methods, (as we pointed out in the first chapter of this section), but by determining and studying the vectors of the electric forces arising during the peaks of cardiac activation. Vector methods of presenting the cardiac forces should not be confused with the study of the electric axis of the heart which represents the position of the mean cardiac vector in a plane corresponding to the frontal plane of the Einthoven triangle. This method is used for the study of the position of the vector in space, and it takes into account its right-left, up-down and forward-backward deviations. This method of studying the electric activity of the heart has come to be known as spatial vectorcardiography.

An outline of the principles of this method of cardiac investigation should consider the following rules (which were partially outlined in the previous chapter) into account:

1. The end result of the heart stimulation (with reference to the ventricles) is the appearance of two major or, rather, mean vectors corresponding to the depolarization and repolarization processes. These vectors are characterized by a definite magnitude and position in space.

2. Any cardiograph lead is a reflection of these spatial vectors, and the nature of the curve in a given lead is determined by the correlation between the lead's axis and the position of these vectors.

3. The amplitude of any deflection in a given lead is determined by the projection of the respective vector onto the lead axis. If the vector direction is perpendicular to the lead axis, there will be no deflection on the EKG, and if the vector is parallel with the lead axis, there will be a maximum deflection. On the whole, it may be said that the amplitude of the electrocardiographic deflection depends on the extent of the vector projection onto the lead axis. /215

4. The polarity of any deflection in a given electrocardiographic lead depends on the manner in which a given vector is projected onto the lead axis. If it is projected onto a positive segment of the lead axis, the EKG will reveal a positive deflection, and if the projection is on a negative segment, the deflection shown on the EKG will be negative.

5. As pointed out in the first chapter of part two, the following equa-

tions are found in electrocardiography:

$$\text{Lead II} = \text{lead I} + \text{lead III} \text{ (Einthoven's Equation)}$$

$$\text{and } aVR + aVL + aVF = 0$$

Moreover, on a six-axis system (see p. 74) the axis of aVF is perpendicular to that of lead I, the axis of aVL is perpendicular to the axis of lead II and the axis of aVR is perpendicular to that of lead III.

The above-cited rules apply to cases when a known vector is used for producing a tracing in some lead. These same rules represent the basic methods of obtaining a spatial cardiac vector from various electrocardiographic leads, that is, they can also be used for spatial vectorcardiography. This calls for leads whose axes are correlated with known spacial planes. In view of these assumptions, Grant and Estes (1951) and Hurst and Woodson (1952) suggested that the spatial disposition of the cardiac vectors be determined in the frontal and anterior-posterior planes of the body, bearing in mind that the axis of the bipolar and single-pole limb leads are localized on the frontal plane, and the axes of the precordial leads are localized on the horizontal plane.

II. METHODS OF SPATIAL VECTORCARDIOGRAPHY

The first step is to determine the position of the vector in the frontal plane by use of a six-axis system. It is preferable to measure the area of the tracing rather than its maximum amplitude. As indicated in Fig. 81, the average vector in lead III is zero, as the QRS complex consists of R and /216 S waves which have an equal area or magnitude. This justifies the conclusion that the direction of the mean vector is perpendicular to that of the axis of lead III. The QRS complex in leads I and II are now positive and their area is almost the same; that means that the mean vector on a six-axis system is directed downward. It is clear in this case that aVR will reveal a maximum negative deflection while aVL and aVF will show small and almost equal positive deflections.

Let us take another example. As indicated in Fig. 82, the QRS area in aVR is almost equal to zero. Hence the QRS vector should be relatively perpendicular to the axis of aVR and directed downward, since the QRS complex in lead I consists of a maximum negative deflection, and the QRS area in lead III is positive. Here the QRS vector should be somewhat deflected toward the axis of aVF since the S wave in aVR is somewhat larger than the area of the R wave. This deflection should be insignificant since the QRS area in lead II is only to an insignificant degree larger than the QRS area in lead I and, further the QRS area in aVL is almost equal to the QRS area in aVF. The /217 range of the vector localization is determined by the generally accepted system.

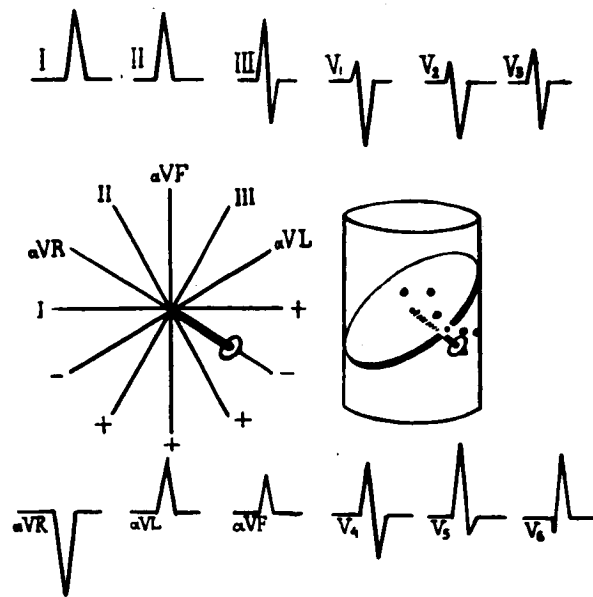


Fig. 81: Deriving a spatial QRS vector from 12 EKG leads (in a diagrammatic representation). The QRS vector is localized in the $+30^{\circ}$ sector, directed downward, to the left and 40° backward (explanation found in the text).

The six limb leads can thus be used to determine the position of the mean vector on the frontal plane. Hurst and Woodson note that the proper methods of such a determination may produce a very accurate picture of the actual vector position with a possible $\pm 5^{\circ}$ margin of error.

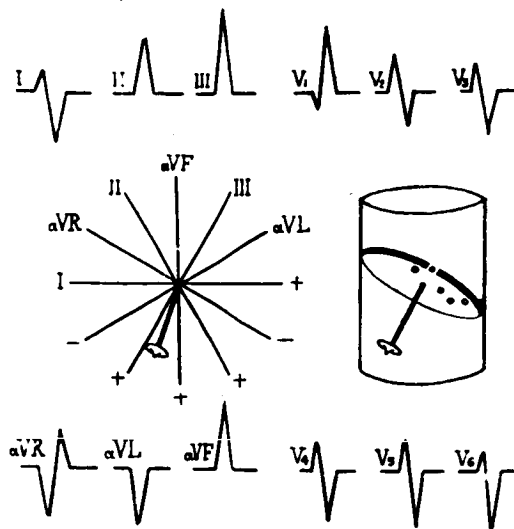


Fig. 82: Deriving a spatial QRS loop from 12 EKG leads (in a diagrammatic representation). The QRS vector is localized in the $+109^{\circ}$ range, directed downward, to the right and 10° forward (explanation found in the text).

The next step is to determine the position of the vector in a horizontal plane. The chest is conditionally presented in the form of a cylinder on which the localization points of the precordial electrode and the electrical center of the heart are indicated (see p. 167). The direction of the mean axis of the heart will then be perpendicular to the axis of the precordial lead where the transitional type of QRS complex is recorded, that is the area of the complex equals zero. Let us take our first example (Fig. 81). Located inside the cylinder is the mean vector, already determined by the limb leads, which originate from the electrical center. The EKG reveals that a transitional complex is being recorded in the precordial V_4 lead. The axis of the given lead is determined, and the vector

deflected in such a way that its direction is perpendicular to that of the given plane. In the second example (Fig. 82), the transitional type of the QRS complex is recorded in V_2 ; that means that the vector will deflect forward in such a way that its direction is perpendicular to that of the plane which passes through the axis of V_2 .

The following tentative average data for the various forms of the chest leads may be used as a basis for a quantitative determination of the extent of the backward or forward vector deflection: the axis of V_1 is deflected /218

from the sagittal plane (which passes through the electrical center of the heart and is perpendicular to the frontal plane) by 10° , the point of its axis is located between V_1 and V_2 , is perpendicular to the frontal plane.

The axis of V_2 is deflected from the sagittal plane by 10° , the plane between leads V_2 and V_3 is deflected from it by 15° . The axis of V_3 by 20° , the plane between V_3 and V_4 by 30° . The axis of V_4 by 40° , the plane of the point between the V_4 and V_5 leads by 50° , the axis of V_5 by $60-70^\circ$, the plane of the point between V_5 and V_6 by 75° , and the plane of V_6 by 80° .

If the vector on the frontal plane is directed to the left, the deflection on the horizontal plane will be backward (except when a transitional complex is found in V_1); but if the vector on the frontal plane is directed to

the right, the deflection on the horizontal plane will be forward (an exception to this rule is again found in V_1). A knowledge of these general rules

makes it possible to determine the vector deflection without the drawing of special cylindrical figures.

The possible margin of error involved in determining the position of the cardiac vector on a horizontal plane is $\pm 15^\circ$, that is three times as great as in the case of its position on a frontal plane. Hurst and Woodson (1952) attribute this to the use of a standard geometrical, cylindrical figure

in all cases, whereas the shape of the chest varies with different people. Besides, the possibility of technical errors involved in the positioning of a chest electrode is fairly large, and the slightest difference of the electrode position from its proper location may alter the picture of the resulting curve considerably.

The above-described method can be used to determine the vectors of the initial and terminal 0.04 sec. The QRS complex which assumes a special significance in the case of bundle branch blocks and infarction can be used also to establish the mean T and RS-T vectors. It should be pointed out that the definition of the T vector, particularly in a horizontal plane, is frequently connected with certain difficulties since the transitional type of deflection is occasionally not recorded within the six usual precordial leads. In such cases, we should either record the additional precordial leads or, which is done more frequently, determine theoretically the approximate localization of the transitional zone. Thus, if the T-wave amplitude gradually increases from V_1 to V_6 , it means that the transitional zone is

located in the right precordial lead V_3R or V_4R . It also means that when the picture is reversed, the transitional zone is farther from lead V_6 . It /219 is interesting to point out, in this connection, that Dal' and Simonson (1953) record, not six, but 34 precordial leads in order to insure greater accuracy.

Zao, Herrmann and Hejtmancik (1957 and 1958) developed another method of studying the main vector of the heart. They used "polarity circles" instead of the six-axis system and the cylinder. Each circle consists of two halves: a positive semicircle (white) and negative semicircle (black). The transitional portions between the semicircles correspond to the zero-potential zones, and the line connecting the two transitional points of one circle is perpendicular to the axis of the given lead. Such a circle is made up for each lead in accordance with its axis and polarity, and the circles arranged frontally from the inside to the outside correspond to leads I, II, III, aVR, aVL and aVF, and those arranged horizontally correspond to leads $V_1, 2, 3, 4, 5, 6$. The rules governing the derivation of a vector are the same

as those by the previous method. The position of the vector is determined by means of a graduated circle: its upper half shows from 0° to -180° , and the lower half from 0° to $+180^\circ$. The circle is divided into twenty-four 15° sections.

Either one of the two methods can be used to find out that the mean QRS and T vectors, which in the normal are directed to the left and downward - the T vector frequently running in parallel with the frontal plane or facing forward or backward, and the QRS vector being oriented backward. There is a certain angle of divergence between the QRS and T vectors. (For normal indicators see p. 363.)

The three main ventricular activation vectors that have been adequately

investigated by Penalzoa and Tranchesi (1955) on a chest model, can be determined in addition to the mean vector. According to these authors, the first, or septal, vector is directed forward and to the right; the second, or left ventricular, vector is directed to the left, downward or backward; and the third, or basal vector is oriented backward to the right and upward. These vectors occur at 0.010 sec., 0.040 sec. and 0.064 sec., respectively, after the beginning of the QRS complex.

It should be pointed out that the method of vectorcardiography is characterized by a number of shortcomings which are due primarily to the fact that the shape of the organism does not even resemble the geometrical figure of a cylinder, and it is far from homogeneous. The cardiac dipole is not in the center of this volumetric conductor either. The shortcomings involved in this method are also characteristic of electrocardiography and vector cardiography.

III. VECTORCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE ELECTROCARDIOLOGIC COMPLEX

The method of spatial vectorcardiography can be used to study any electrocardiographic phenomenon. But from a practical point of view, this method of analysis is definitely important for the study of hypertrophy, bundle branch blocks and myocardial infarctions. The following is a brief description of vectorcardiographic deflections in such conditions /220

1. Ventricular Hypertrophy

In the case of hypertrophy, the mean QRS vector faces the hypertrophied section of the heart, and the RS-T and T vectors are characterized by a reverse direction.

The QRS vector of a hypertrophied left ventricle (Fig. 83) is directed more to the left, downward and backward, and the T vector is frequently directed to the right and forward.

The mean QRS vector of a hypertrophied right ventricle (Fig. 84) is directed to the right and forward, and the T vector is oriented to the left and somewhat backward.

Ventricular hypertrophy is accompanied by an increasing angle of divergence between the QRS and T vectors until they acquire opposite directions. Moreover, an RS-T vector with the same direction as a T vector also comes into view. /221

2. Bundle Branch Block

It is a known fact that the depolarization of the ventricles begins from the septum and moves to their endocardial surfaces. The time gap between these two moments of cardiac stimulation is 0.04 sec. In the case

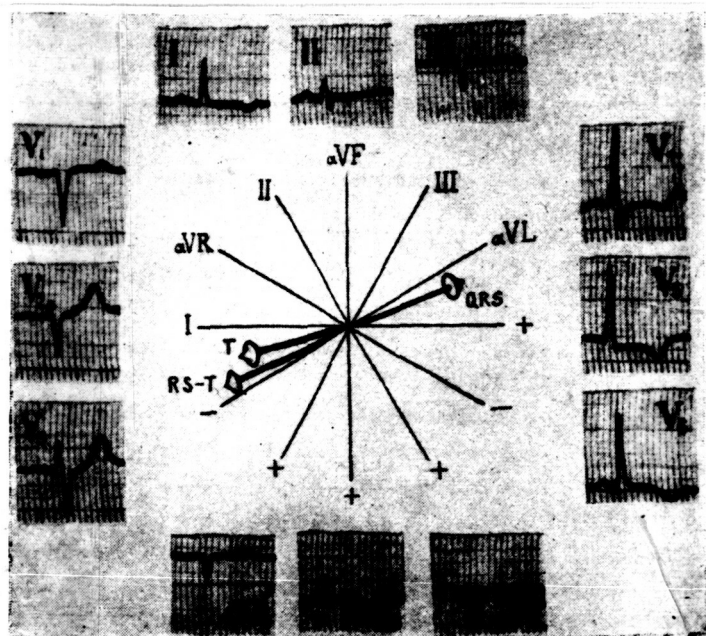


Fig. 83: Left ventricular hypertrophy. Electrocardiogram of patient, P. V., age 49, with the following diagnosis: hypertension, stage IIb. The QRS vector is directed to the left and backward by 30° (transitional complex between leads V_3 and V_4); the T vector is directed to the right and forward by 30° (transitional complex between leads V_3 and V_4); in this case there is also an RS-T vector whose direction is parallel to that of the T vector.

of blocks during the initial 0.04 sec., the electric forces originate only in the unaffected ventricle; they are eventually joined by the forces of the affected ventricle, and the vector of the final 0.04 sec. is oriented in its direction.

In the case of a blocked left branch (Fig. 85), the electric forces are of a right ventricular origin in the first 0.04 sec., and the QRS vector is oriented to the right and forward. Eventually, with the onset of the left ventricular forces, the vector becomes oriented more to the left and backward. Thus, in the case of a left bundle branch block, the direction of the initial vector is the same as under normal conditions, whereas the terminal vector usually occupies the upper left quadrant in a Cartesian coordinate system.

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In the case of a right bundle block (Fig. 86), the electric forces originate only in the left ventricle, and the vector is directed to the left and somewhat backward.

A bundle block increases the angle of divergence between the QRS and T vectors to 180° .

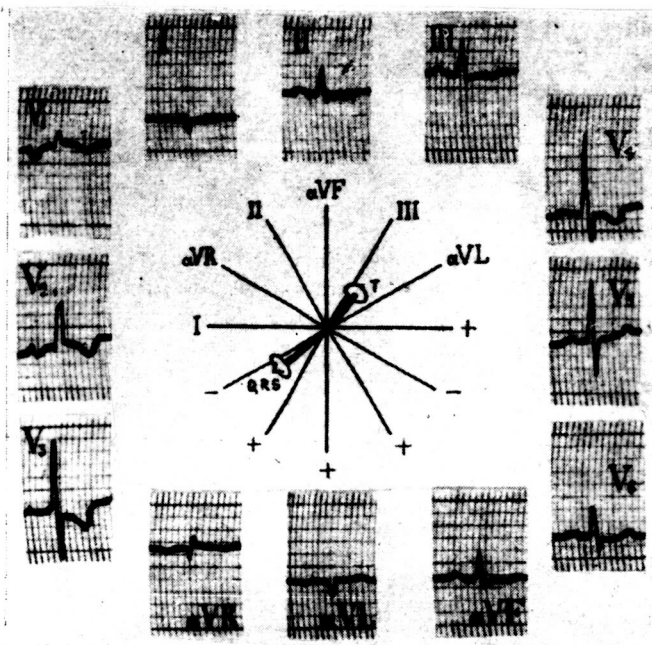


Fig. 84: Right ventricular hypertrophy. The EKG of patient, S. Zh., age 19, with following diagnosis: mitral defect with prevalent stenosis. QRS vector directed to the right and forward by 20° (transitional complex in lead V_3), the T vector is directed to the left and backward by 50° (transitional complex between leads V_4 and V_5).

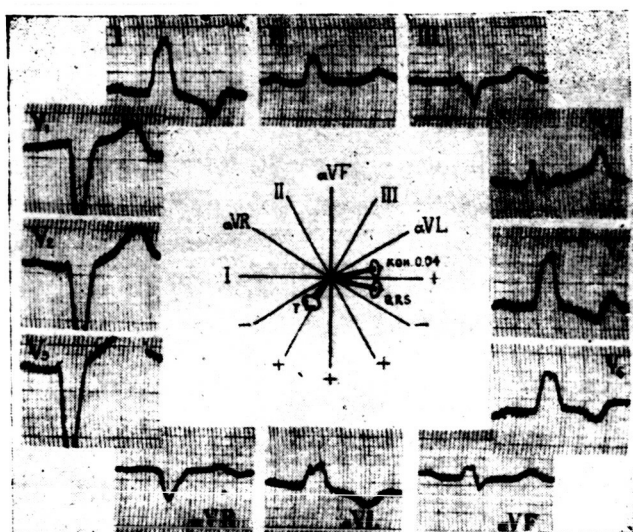


Fig. 85: Left bundle branch block. An EKG of patient V. A., age 36, with /222 following diagnosis: hypertension, stage Ib. The QRS vector is directed to the left and backward by 30° (transitional complex between leads V_3 and V_4), the vector of the final 0.04 sec. is directed to the left and backward by 30° (transitional zone V_3 - V_4), the T vector is directed to the right and forward by 60 - 70° (transitional zone V_5).

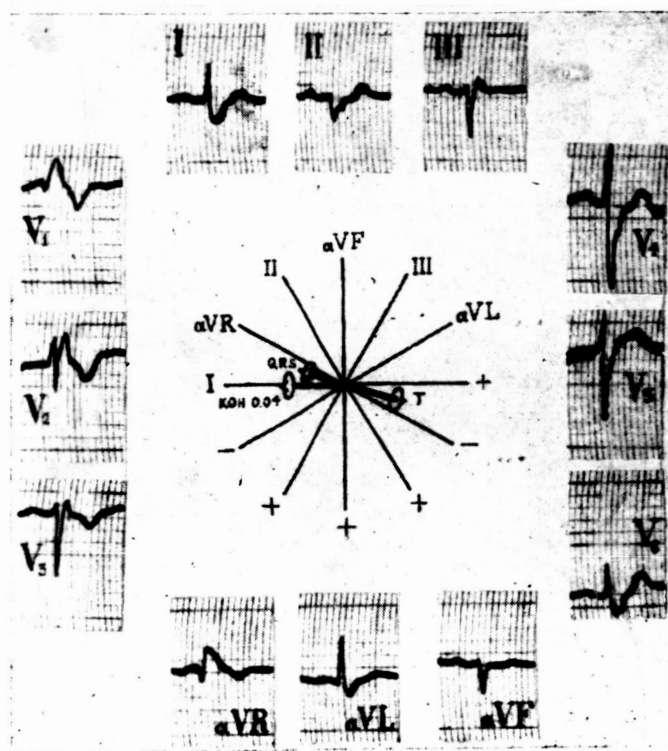


Fig. 86: Right bundle branch block. An EKG of patient K. A., age 42, with the following diagnosis: hypertension, stage IIa. QRS vector directed to the right and forward by 15° (transitional zone between leads V_2 and V_3).

The vector of the final 0.04 sec. is directed to the right and forward by 30° (transitional zone V_3 - V_4), and the T vector is directed to the left and backward by 30° (transitional zone between leads V_3 and V_4).

3. Coronary Insufficiency

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We do not think it necessary to dwell on the characteristic changes produced on a vector EKG by various localizations of an infarction. Suffice it to say that the vector of the initial 0.04 sec. of an infarct and the T vector are turned further than that of the QRS vector, and in a direction opposite to it. This is accompanied by the emergence of an RS-T vector which, conversely, faces the infarcted area. This is illustrated in Fig. 87 by a vector EKG of a patient suffering from an anterior wall infarct.

IV. CONCLUSION

The method of spatial vector electrocardiography is a good method of training the thinking of the electrocardiologist, and acquiring the habit of a quantitative and graphic presentation of the mechanism governing the cardiac contraction. It facilitates the mastery of the vector principles, and the appropriate methods and terminology. Its importance in the train-

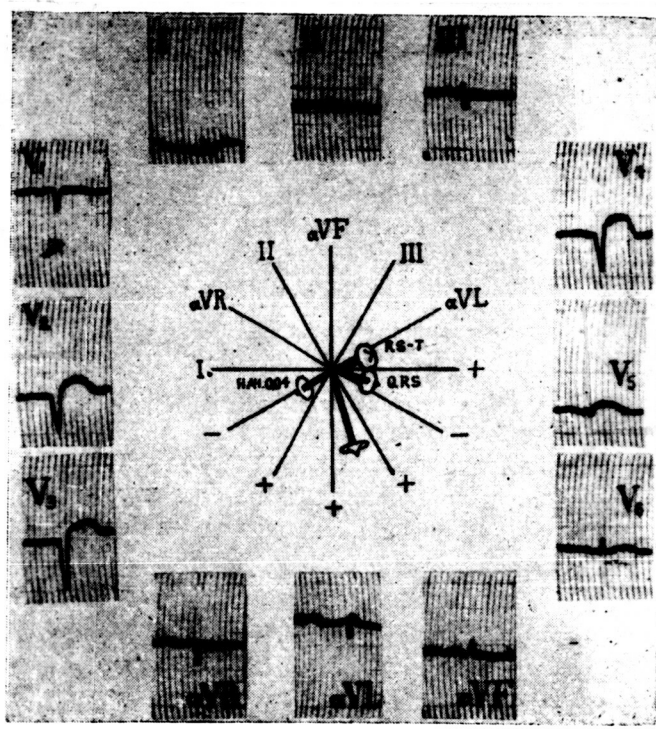


Fig. 87: Anterior infarction. A An EKG of patient E. G., age 48, with following diagnosis: acute anterior infarction, and atherosclerotic heart disease. QRS vector is directed to the left and backward by 75° (transitional zone between leads V_5 and V_6), the vector of the initial 0.04 sec. is directed to the right and backward by 75° (transitional zone V_5 - V_6). The RS-T vector is directed to the left and forward by 80° (transitional zone in lead V_5), and the T vector is directed downward and is parallel with the frontal plane (transitional zone V_1 - V_2).

ing of beginners in the field of electrocardiology should also be emphasized.

The variations of this method provide convincing proof of the correctness of the theoretical foundation of electrocardiography. The various forms of the complexes are not a result of accidental phenomena, but are a reflection of objective laws. The use of the principles of spatial vectorcardiography in our practice has contributed a great deal to a thorough understanding and more correct interpretation of the different physiological and pathological EKG variations.

It should be pointed out that a simultaneous vectorcardiographic examination obviates the necessity for a standard electrocardiographic analysis, as the information yielded by a vectorcardiographic examination is relatively better. This method of analysis is very important whenever it is impossible to record a vectorcardiogram.

PART THREE

CLINICAL STUDY OF MECHANICAL ACTIVITY OF THE HEART

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BALLISTOCARDIOGRAPHY

I. INTRODUCTION

The ballistocardiographic method can be used to study the condition of the heart by recording the recoil movements of the body resulting from the expulsion of the blood from the heart and its flow along the circulatory system. Gordon discovered the above mentioned movements as far back as 1877, after observing the rhythmic oscillations of a scale indicator which were synchronous with the pulse of the test subject on the scale. He subsequently proposed the use of a special suspended table for recording these movements. A little later, in 1881, Landois designed a verticle apparatus based on the principle of the Gordon device. By using a special horizontal table in 1905, Henderson obtained an "efficiency curve" and developed a formula for the quantitative study of the cardiac stroke volume. Eventually many authors proposed a variety of original designs, such as the Heald and Tucker suspension system (1922), the Angenheister and Lau seismograph (1928) and a chair with a spring mechanism, the Abramson cardiodynamograph (1933).

The scientific refinements applied to ballistocardiography, the development of its theoretical and practical aspects and its clinical application actually began with the well-known studies made by Starr and his colleagues in 1939. Even the term "ballistocardiography" itself was coined by Starr. It derives from the Latin word "ballista" which means to throw. ("Ballista" was the name of the weapon used in the old times to throw heavy objects onto the enemy's positions; thus, the term "ballistics", the science of the movements and trajectories of shells and rockets was derived.)

In the Soviet Union the development of the ballistocardiographic method was furthered by the first studies made by V. V. Parin and his colleagues in 1954 who worked on the problems affecting the techniques and clinical aspects of that method. The very rapid popularization and application of ballistocardiography in the Soviet clinics may be judged from the fact that it was considered possible to convene the First All-Union Conference on Ballistocardiography in Moscow as far back as 1959.

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Dynamocardiography, a method designed to study the mechanical phenomena produced in the circulatory system by cardiac contractions, has also undergone successful development in the Soviet Union in recent years. The method requires a relatively complicated device, but its inclusion in a five-channel electrocardiograph (BDKG-01) as well as its production as a separate complete system will make it more popular. Dynamocardiography can be used to record longitudinal and lateral curves and study the cardiac forces in various axes and planes. The authors of that method, Ye. B. Babskiy and V. L. Karpman (1958), find that there is a big difference between the two methods from a physical point of view. Ballistocardiography records the body displacement, while dynamocardiography regis-

ters the shifting center of gravity in the chest. Since these systems record different manifestations of the cardiac activity (V. S. Gurfinkel' and T. S. Vinogradova, 1959), there is no temporal correlation between the various characteristic phases of these curves.

Kinetocardiography has also been successfully applied recently (Edleman et al., 1953; L. B. Andreyev, 1961; I. Ye. Oranskiy, 1961). The data unit is placed directly over the cardiac area, and it records the local movements of the thoracic wall during cardiac contractions.

II. TECHNIQUES OF BALLISTOCARDIOGRAPHY

1. Physical Principles

As pointed out by Dock, Mandelbaum and Mandelbaum (1953), the heart and the major blood vessels are suspended from the skeleton by cords made of connective tissue which function as springs. These cords are located primarily between the major parts of the cardiovascular system and the bony skeleton. Then there is contact between the skeleton and the surface of the table on which the body of the test subject has been placed. Every cardiac contraction produces ballistic forces whose nature is determined by the strength of the cardiac contractions, by the condition of the circulatory system as a whole, and by the elastic properties of the various soft tissues of the organism. These forces differ as to the time of their origin as well as by their magnitude and direction. An integral summation of these forces produces one common effect: a displacement of the body's gravitational center. This is accompanied by a certain movement of the body in a longitudinal, lateral or vertical direction. These movements are made by the skeleton of the organism. The mechanical forces generated by cardiac contractions and blood circulation are transmitted through the connective-tissue connections to the hard osseous foundations of the body, and the latter produces these movements in relation to the immovable surface on which the test subject rests. These forces undergo a considerable qualitative change as they pass through the above mentioned connections. That means that the seismic movements of the skeleton are quantitatively or qualitatively not quite equal to the cardiovascular forces, but they merely reflect their general dynamics. Yu. A. Vlasov (1961) justly notes that the pulse is transmitted from the point of generation to the recording instrument through a number of nonhomogeneous systems in which the real nature of the pulse is considerably distorted. We agree with R. M. Bayevskiy (1962) that the following factors which change the true nature of the cardiovascular forces on a seismic ballistocardiogram are of major importance: elastic output and inhibition forces, natural oscillations of the body whose frequency characteristics are very similar to those of the cardiovascular forces, and the interference of all these various forces. We believe that the condition of the above-mentioned spring-like mechanisms deserves a great deal of attention. We know that a high degree of stress may be reflected on a ballistocardiogram by the appearance of pathological changes resulting from the changes in the musculoskeletal system alone (Hakim et al., 1957).

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The mechanical movements of the body, like any other mechanical movement, have three parameters: displacement, speed and acceleration. There is a definite correlation between these parameters, and one parameter can be derived from another one by an appropriate mathematical operation. The physical relationship between the ballistocardiograms of displacement, speed and acceleration may be described as follows. A speed ballistocardiogram is a derivative of a displacement ballistocardiogram, and an acceleration ballistocardiogram is a derivative of a speed ballistocardiogram (the derivative is a concept of differential calculus indicating the speed of a changing function).

The ballistic forces generated by dynamic cardiac action has magnitude and direction, that is, they are vectorial forces. It is from this point of view that the attempts of individual authors to develop a method of spatial vector ballistocardiography can be understood (M. I. Tumanovskiy and Yu. D. Safonov, 1957; V. L. Karpman, 1959; Braunstein, 1954). In day-to-day work it is practical to record these movements in one definite direction. It is possible for example, to record the lateral vectors and to obtain a lateral ballistocardiogram. The ballistocardiogram recorded in actual practice is most frequently longitudinal, as the longitudinal forces are the most pronounced (they are three times as strong as the lateral forces and six times as strong as the vertical forces), and are relatively simpler to record from a technical point of view. /230

2. Techniques

The system used in any ballistocardiograph is designed to detect and record the ballistic cardiovascular forces arising in the course of the cardiac cycle. That means that the device should be designed to possess a mechanism that records the body movements of the test subject, a converter to change these mechanical forces to electrical, and an amplifier and recording device to amplify and record the electrical phenomena coming from the converter. The design should include an electrical filter capable of passing or blocking the alternating current of a certain frequency band reaching its input.

Although all existing ballistocardiographs consist of the above-described components, they are divided into two large groups direct and indirect devices, according to the manner in which they measure the mechanical forces of the body.

In the case of indirect ballistocardiography, the test subject lies, sits or stands on a movable surface (table, chair or platform), and what the apparatus records is not the body movements, but the movements of the movable surface caused by the body movement. The resulting curve represents the totality of the forces acting in the organism; and it is affected by such factors as the frequency characteristic of the surface, the degree of its mobility, the weight of the test subject, etc.

Direct ballistocardiography records the movements of the body occurring

in any of its parts. In such cases the test subject is placed on an absolutely motionless surface, and the resulting curve depends entirely on the seismic movements of a given area of the organism. This method is technically considerably simpler, and it is free of a number of moments that tend to complicate the indirect method.

Electrical data units are used in modern ballistocardiography; they may be photoelectric, piezoelectric, electromagnetic, capillary units, etc. The selection of a data unit depends in some measure on the parameter of the body movement to be recorded. The electromagnetic data unit is used most in practical ballistocardiography as it records a better speed curve. /231

The devices used in modern electrophysiology, particularly electrocardiographs, can be used as recording devices; their frequency includes the range of the ballistocardiographic phenomena, and their sensitivity is more than adequate. We use the multichannel apparatus and record a number of other indices of cardiac mechanism in addition to a ballistocardiogram.

3. Indirect-Method Ballistocardiographs

In 1939 Starr designed his first high-frequency apparatus. A special table is suspended from the ceiling by four steel cables in such a way that it can move only along its longitudinal axis. A special spring with a small mirror on top of it is attached to one end of the table. The table movements produced by the ballistic forces of the test subject's heart beat rotate the mirror. These rotations are registered by a recording device. The total weight of the table is about 50 kilograms. There is no mechanical damping in this system; such damping is performed by the test subject's body.

The modern models of the high-frequency Starr devices consist of a bulky table with a fixed base and a movable surface connected to it by hard springs. The damping method is not applied in this system either. The table also has a high natural frequency.

Nickerson and Curtis (1944) propose the use of a critically damped low-frequency table for two reasons. First, they find that the table must be damped by a device that does not produce any friction, and since excessive or insufficient damping may cause certain changes, the entire table should be critically damped. Secondly, they believe that the picture of a ballistocardiogram recorded by low-frequency tables depends almost entirely on the elastic tissue properties, and low-frequency tables with less than two cycles per second (between 1.0 and 1.5 cycles per second) should therefore be used. Rappaport, Sprague and Thompson (1953) find that such a table produces a good record of the high frequency components of the ballistic forces.

Reeves et al. (1957) describe a low-frequency acceleration ballistocardiograph built on the principle of a horizontal pendulum, whereby the test subject's shoulders are fastened to the table. Taylor and Walker (1957) designed a device that can record both a high-frequency and critically damped

low-frequency curve. Scarborough et al. (1958), propose an ultra low-frequency system whereby the resulting curve conforms well to the various phases of the cardiac cycle. A BDKG-01 type ballistocardiographic attachment to a five-channel electrocardiograph can make a high-frequency recording of the longitudinal movements of the table. /232

4. Direct-Method Ballistocardiographs

The direct method of recording a ballistocardiogram was first clinically applied by Dock and Taubman in 1949. These authors recorded the ballistocardiogram by means of glycerin-filled capsules placed on the head. The pressure changes in the liquid produced by every contraction of the heart were recorded by an appropriate device. They also used a photoelectric method of recording whereby the intensity of the light hitting the photocell changed in accordance with the body movements. This method was used to record a displacement curve. The most widespread method now in use is the "electromagnetic" recording. The data unit is placed on some area of the body, and the induction current produced by the movements of that area is recorded. The data unit may be placed on any part of the subject, but the nature of the resulting curve will be affected somewhat by the particular placement of the unit. Dock, Mandelbaum and Mandelbaum (1953) note that the head and upper extremities are technically unsuitable locations for the data unit as the movements in the upper extremities are relatively slow. The chest has definite advantages, as the longitudinal ballistic forces are good in that area. Actually, however, it would be inexpedient to record a ballistocardiogram of that region, since the data unit resting on the chest would be a technical hindrance to a simultaneous examination of the precordium. Dock suggests that the unit be placed on the distal part of the lower leg in the belief that this procedure is technically quite simple and that the resulting curve differs very little from the one recorded by the indirect Starr method.

This is how the electromagnetic data unit is designed. A specially adapted plank, usually wood, is placed on both shins and fastened with rubber tape. The free space in the middle of the plank contains two small coils, each consisting of a large number (up to 8,000) of turns of thin wire. Between the coils is a small gap (not more than 15 mm) facing the feet, and in that gap is a flat steel magnet fastened to a solid support. The coils are thus located in a magnetic field, and as the body moves in accordance with the physical rules of electromagnetic induction, it generates an induction current whose force is directly proportional to the speed of the body movement. The resulting current is recorded by means of an ordinary electrocardiograph; the resulting curve is a speed ballistocardiogram. The respiratory movements do not produce any noticeable effect on the nature of the resulting curve because of their low frequency. The device is equipped with a 2-microfarad condenser to filter out the interference from muscular tremor. Dock proposes the recording of a so-called "diagnostic" ballistocardiogram, using a 20-microfarad condenser, in daily practice. Such a curve actually represents a combination of displacement and speed parameters. Although this ballistocardiogram cannot be classified as some concrete parameter of movement from a physical point of view, /233

it is very useful in daily practice as it clearly outlines the structure of the complexes and the rhythmic respiratory fluctuations of the curve.

The design used by V. V. Parin and A. V. Mareyev (1954) as well as by A. I. Gefter and his colleagues (1958) is almost analogous to that of the Dock data unit. Biörck and Thorson (1953) propose a special piezoelectric ballistocardiograph. N. S. Fetisov (1960) uses a piezomagnetic data unit in which the body movements are communicated to the piezomagnet through two magnets: one is located on the receiving device on the shins of the test subject, and the other is connected to the piezoelectric cell.

Smith (1952) developed a method of expressing the speed of the body in absolute units. In the Smith data unit a round magnet is located on the plank which is placed on the shins, and inside it is an induction coil with about 100,000 turns of wire fastened to a support. Smith and Bryan (1953) use such a device to record all the three parameters of movement.

G. Ye. Tsintsadze (1957) proposed a device for recording ballistocardiograms of speed, acceleration and displacement consisting primarily of a mechanical system of indirect recording combined with the Smith principle of direct ballistocardiography. The mechanical system of the device is based on the principle of a pendulum with a free suspension. The advantage of the pendulum system is that it can be easily calibrated. Such a design produces a high number of natural oscillations, and reduces the second degree of freedom of movement to a minimum. The author believes that this ballistocardiograph is simple to use, easy to carry and inexpensive.

R. M. Bayevskiy (1959) designed a portable electromagnetic ballistocardiograph based on both indirect and direct recording principles. The device, consisting of two small wooden platforms solidly interconnected by four flat springs, is placed on the shins of the supine patient. A magnet is fastened to the upper platform, and an induction coil to the lower one. The electromagnetic recording principle is used; as the upper platform is shifted in relation to the lower one, the apparatus senses the body movements along the longitudinal axis. This device, according to the author, is characterized by a constant mechanical and electrical sensitivity, it is highly resistant to lateral shifts and twists and it can be used for recording under any conditions, even at the bedside of a gravely ill patient. /234

K. P. Buteyko (1959) proposes a ballistocardiograph of displacement, speed and acceleration with a calibrating device whereby the pendulum, after calibration, is connected to the test subject in the direct-method ballistocardiography, and to the table as in the case of indirect ballistocardiography.

Ballistocardiography is now being used also in experimental investigations. These devices are based on the principle of the electromagnetic data unit; A. N. Fedoseyev (1958) used them on dogs lying on their stomachs, and A. V. Mareyev (1958) suggests that they be applied to subjects lying on their back. A good method is suggested by Frederick et al. (1955): the dog is to be kept in a sand bag in a supine position.

There are numerous recording systems and principles in modern ballistocardiography. Attempts have been made to classify all the available ballistocardiographs on the basis of their different principles, by the way they sense the movements, by the manner they record the movement parameter, by their frequency characteristics, etc. The most complete and all-inclusive classification is proposed by V. V. Parin and R. M. Bayevskiy (1961). They grouped all the systems according to four basic principles:

1. By the number of axes simultaneously recording the mechanical phenomena (a linear, planar and spatial ballistocardiogram).
2. By the physical characteristics of the recorded mechanical phenomena (dynamic and seismic ballistocardiograms).
3. By the recording parameter of movement (a displacement, speed and acceleration ballistocardiogram).
4. By the method the movements are sensed (direct and indirect-method ballistocardiogram).

Under this scheme, kinetocardiography applies to the seismic system, dynamocardiography to the direct dynamic system, and the direct Dock method is a seismic linear system; the indirect dynamic systems include the ultra low-frequency systems.

It may be concluded that the situation here is about the same as in vectorcardiography: a great number of recording systems, and the curves recorded by these systems are not always analogous or comparable. For example, the displacement, acceleration and speed curves recorded by the ultra low-frequency systems are considerably different from one another. Most typical of the ballistocardiograms are the curves recorded by the direct Dock method and indirect Starr method. They are similar in many ways since both of them record the seismic effect of the heart's contraction and they are part of seismocardiography (R. M. Bayevskiy, 1962). This is our approach to this problem. There is no doubt in our mind that a commonly accepted and clinically tested system is necessary. Besides, such a system should not be based strictly on physical indicators. The fact that a curve reflects speed, acceleration or displacement of systole or that a ballistocardiogram is dynamic or seismic is, in the final analysis, of little interest to the biologist. These concepts should interest primarily the physicist or, rather, the biophysicist, but not the cardiologist. We prefer to use the direct Dock method, as it is easily applied from a technical point of view and it produces good results. The data unit can be easily set up, and the minor inaccuracies involved in the adjustment of the various components of that device do not affect the nature of the curve, as the field of the flat magnet makes the electromagnetic data unit relatively insensitive to such changes. It is a known fact that the output voltage of the device is almost constant. Changes are within the range of $\pm 10\%$ if the positions of the magnet and the coils are moved up to 5 mm from the ideal position in the vertical and horizontal planes (Dock, Mandelbaum and Mandelbaum, 1953). True, this device cannot be used in all cases, as for

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example on a bed-ridden gravely ill patient, but it could be used if such patients were placed on specially adapted beds beforehand.

We do not want to deny the definite advantages of the indirect systems, such as the Starr and Nickerson systems, and we have recently begun to use a high-frequency ballistocardiographic table with a five-channel electrocardiograph. These devices are bulky, technically complicated and require a special room. Besides, it should also be borne in mind that the wave amplitude of an indirect ballistocardiogram is determined by the body weight, and its tracing may therefore change in accordance with the test subject's body weight and subcutaneous fatty layer. We do not agree with Haas and Clensh (1957) who claim that the Dock method has no scientific - research significance, and that it cannot even determine the qualitative aspect of a ballistocardiogram. True, in the case of the direct method, the physical properties of the subject tend to exaggerate some aspects of the internal forces and minimize others, thereby distorting the curves. The quality of the curves can be improved if the patients are fastened to the table more securely (Nurdergraaf, 1961). We agree with Dock, Mandelbaum and Mandelbaum (1953) that the direct method made it possible to take ballisto- /236 cardiography out of the laboratory and introduce it in clinical practice on a wide scale.

We will not dwell on the physical and mathematical bases of ballistocardiography which have already been elaborated by Buerger and his colleagues (1956, 1957 and 1961), as well as by R. I. Gismatulin (1959), etc. These problems are only briefly dealt with in various parts of this chapter.

5. Methods

The use of the direct method calls for conditions whereby extraneous mechanical movements, however insignificant, could not affect the recording of the curve. The examination room should be far from the physical therapy department, and the areas containing motors, elevators, etc. The quality of the table is very important. It should be sturdy and rigidly fixed to a solid floor that does not reflect even insignificant vibrations from various parts of the building. We have found a successful solution to this problem by the use of legless tables which do not come in contact with the floor; they rest on two metal supports which are built into a heavy structural wall of the room. The use of such tables makes it possible to record very accurate curves, and every test made by Dock, Mandelbaum and Mandelbaum (1953) which determined the quality of the table produced good results (a ballistocardiogram of a patient recorded on such a table and on the floor should produce an identical picture, and the wave amplitude should not vary by more than 10-15%).

When the direct method is used, the shins should be raised 6-8 cm above the table surface by placing a sand bag or block of wood under the Achilles tendons in order to exclude any accidental movements in the crural joints and reduce the loss of the ballistic forces generated by the friction between the tissues of the subject and the table. The wooden block should be rigidly fixed, the movements of the data units and the extremity must

be fully integrated.

The proper preparation of the test subject for a ballistocardiographic examination is very important. Such preparations are, of course, required for any examination but they are particularly important in ballistocardiography. The patient must be in a condition for basal metabolism, and should refrain from smoking and physical exertion before the examination. He must relax on the table for 10-15 minutes before the ballistocardiogram recording. He must be kept comfortable with a pillow under the head and a bedsheet or soft mattress on the table extending to the upper part of the hips. The principle of the examination should have been explained to the patient, as his attitude toward it is very important. Patients in a state of unconsciousness and children do not submit to such an examination. It should be pointed out also that a ballistocardiogram cannot be recorded in the case of tremor, tachycardia or dyspnea. In the course of the examination the patients must be able to breathe freely, and without effort, his abdomen should not be under strain. The weight, height and age of the patients must be written down. The actual recording should be accompanied by calm breathing, and at least 12-13 complexes should be traced. The next step is to record 4-5 complexes with the patient holding his breath during inspiration and during expiration (with an open mouth, to avoid the Valsalva effect).

The problem of standard calibration in ballistocardiography is fairly difficult. According to the principles proposed by Starr for the indirect method, the table spring should be regulated in such a way that a 280-gram force produces a 10 mm stylus shift. We have been using an electromagnetic Dock data unit and by controlling the amplification of the recording device in such a way that 1 millivolt produces a 10 mm stylus shift. As recommended by the American Heart Association, the standard parameters of the movements of a person of average weight and size in one centimeter of recording should be: displacement 0.006 cm, speed 0.1 cm/sec., acceleration 2 cm/sec². Dock, Mandelbaum and Mandelbaum (1953) propose the use of a pendulum to determine the speed produced on a curve by a deflection corresponding to one millivolt.

The polarity of the connections should be checked before the examination. The polarity should cause the caudal ballistic forces to produce a downward deflection on the ballistocardiogram, and the cranial forces an upward deflection. The method of determining this is very simple: a tap on the shoulder should produce a downward stylus movement, and a tap on the foot an upward stylus movement.

A ballistocardiographic examination should be accompanied by an electrocardiographic one. The simultaneous recording of a phonocardiogram and arterial sphygmogram produces even more information. This calls for a multi-channel apparatus. If such a device is not available, an ordinary single-channel electrocardiograph may be used, but the R waves of the electrocardiogram should be superimposed on the ballistocardiogram by the use of a special system of connections.

The resultant sum of all the similar or counter-action, synergistic or antagonistic ballistic forces originated by cardiac contractions is expressed by a number of successive waves which, as proposed by Starr and recommended by the American committee on ballistocardiographic terminology (1953), are conditionally designated as H, I, J, K, L, M and N (Fig. 88). Of these, the H, J, L and N waves reflect the body movements in a cranial direction and are considered positive waves, and the I, K and M waves reflect the body movements in a caudal direction and are looked upon as negative waves. The H wave is, in a number of cases preceded by a presystolic G wave and the N wave is followed by an O wave. Unlike the electrocardiogram, the ballistocardiogram has no segments which occasionally complicate the determination of the position of the isoelectric line. Moreover, the ballistocardiographic waves have smooth contours without sharp peaks, and their width, amplitude and to some extent their configuration are not confined to narrow limits and fluctuate widely within the norm.

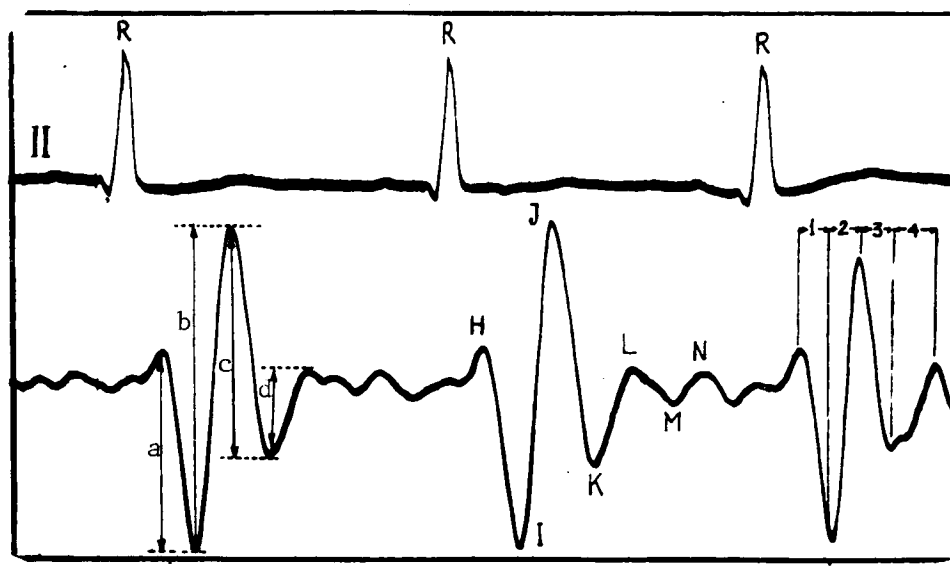


Fig. 88. A normal ballistocardiogram. On the first channel the electrocardiogram shows lead II. On the second channel is a ballistocardiogram: wave amplitudes (a) HI, (b) IJ, (c) JK and (d) KL; the duration of the intervals is (1) H-I, (2) I-J, (3) J-K and (4) K-L (wave I of the patient under consideration is a little deep).

Wave J on the ballistocardiogram has the highest amplitude, and may be considered as a standard when comparing the magnitudes of the other wave in relation to its amplitude. /239

The H wave usually has $1/4$ the amplitude of the J wave, but it may be somewhat larger or smaller. The amplitude of the H wave usually increases during the expiration. This wave is the most variable component of a normal

ballistocardiogram. The I wave is usually larger than the H wave, and its amplitude is approximately 1/2 that of the J wave, and fluctuates a great deal in the case of healthy people.

The K wave resembles the I wave but it is characterized by a steep rise, and its amplitude is usually a little greater than that of the I wave. That wave is reduced by inspiration and somewhat increased by expiration. S. S. Belousov (1958) notes that during inspiration the K wave amplitude amounts to 67% of the J wave magnitude, and during expiration it increases to 73%.

The L, M and N waves may sometimes be missing, particularly in the case of tachycardia or when faulty recording devices are used. The L wave amounts to 64% of the J wave amplitude (S. S. Belousov, 1958), although it is frequently smaller than the H wave (Brown et al., 1952). The M wave has about the same configuration as the L wave but with caudal direction. The N wave also resembles the L wave, and its amplitude may be 1/2 that of the J wave.

All these ballistocardiographic waves may be grouped into two complexes: the HIJK or systolic complex, and the LMN or diastolic complex. It is also called the post-systolic complex. It should be borne in mind that the point under discussion is the mechanical or, rather, hemodynamic systole of the ventricles. The atrial systole is not reflected on the ballistocardiogram as its wave complex becomes stratified on the larger ventricular complex. The H wave is occasionally preceded by undetermined waves (F and G waves) which may occasionally be attributed to an atrial systole. In some cases a pronounced retardation of the atrioventricular conduction or a complete atrioventricular block is accompanied by the appearance of atrial systolic waves which have largely the same configuration and picture as the corresponding waves of the ventricular complex but with a lower amplitude. They may be designated by the lower case letters h, i, j and k, with interval P-h = 0.10 sec., P-i = 0.16 sec. and P-j = 0.24 sec. (Nickerson, 1949).

The origin of the ballistocardiogram waves is linked to the ballistic forces generated by the individual cardiac contractions. The genesis of any wave involves the participation of a number of physiological mechanisms, but only one of these becomes a determining factor. The genesis of the waves can be well represented by recalling the phasic structure of the cardiac contraction (see p. 34) as well as the correlation between the individual ballistocardiogram waves and the structure of the other well known indicators of cardiac contraction. The systolic complex of the ballistocardiogram begins on an average of 0.07-0.09 sec. after the electrical excitation of the ventricles, and ends after the completion of the T wave. The H wave coincides with the beginning of the apical impulse curve. The I wave appears after the I sound on the phonocardiogram, usually in the middle of the I-II interval. The J wave takes shape in the interval between the peak of the percussive wave and the incisura of the carotid artery sphygmogram, and the K wave almost coincides with the vibrations of the 2nd sound on the phonocardiogram. The L, M and N waves are generated in the course of the T-P segment of the electrocardiogram (see appropriate chapters for the values of these indicators).

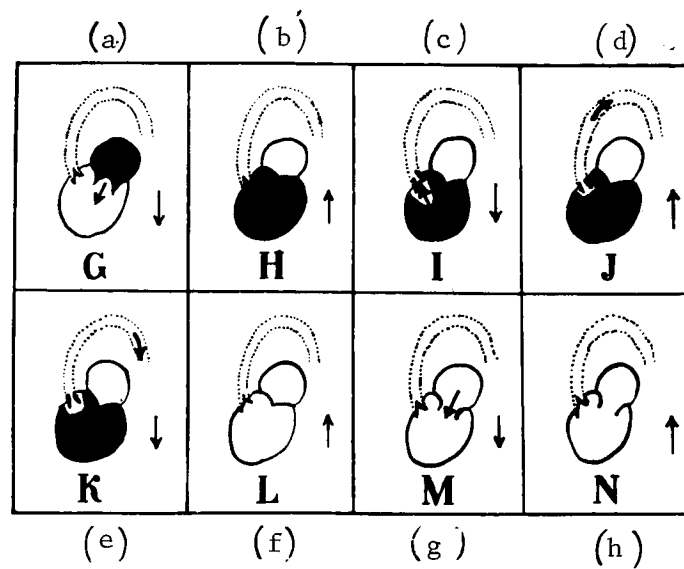


Fig. 89. Diagram of the mechanisms governing the generation of the ballistocardiographic forces. The arrows inside the heart or vessel (aorta) indicate the direction of the blood flow, and the arrows on the side show the direction of the ballistic forces. The left atrium and left ventricle are shown in the diagram; the shaded portion is in the systolic phase.

Shown in Fig. 89 is the diagram of the mechanisms governing the origin of the waves. As the atria contract, the blood rushes from them into the ventricles producing forces in a caudal direction. These actions may cause the appearance of the G wave. With the onset of the ventricular systole tension phase, the atrioventricular septum is pushed upward; the result of this and, to some extent, the apical impulse is the emergence of forces with a cranial direction causing the formation of the H wave. The semilunar valves open in the beginning of the rapid expulsion phase of ventricular systole, and the blood flows out of the ventricles into the major blood vessels. The recoil forces generated in this process pull the heart and the root of the aorta in a caudal direction and produce wave I. In the middle of the rapid expulsion phase the pulse wave and blood current impinges against the arch of the aorta and bifurcation of the pulmonary artery thereby generating forces in a cranial direction; the result is wave J. By the end of the expulsion phase, the pulse wave and blood current reach the level of the abdominal aorta, and impinges against the bifurcation of the aorta. The gradual reduction in the speed of the blood flow in the thoracic and abdominal portions of the aorta result in the generation of caudal ballistic forces which cause the emergence of the K wave. /241

During the isometric relaxation phase, the closing of the semilunar valves compresses the atrioventricular septum into the atria pushing the mass of blood inside them upward; the result is wave L. The atrioventricular valves open early in the period of ventricular filling, and the blood from the atria rushes into the ventricles. The caudal forces generated in the process produce wave M. In the late period of the diastolic blood in-

flow, the pressure in the ventricle is higher than that in the atria, and the flow of blood into the ventricles comes to a stop; the result is a reverse current into the atria and the generation of forces in a cranial direction. This is how wave N comes into being. We believe that the origin of diastolic waves should not be attributed to ventricular filling alone, as in that period of the cardiac cycle involving as it does, low intracavitary and intravascular pressure, the formation of any pronounced ballistic force is impossible. The experimental observations made by Thomas et al. (1956) show that these diastolic waves very probably reflect the real cardiovascular forces. An important factor in the origin of these waves also is peripheral vessel filling, as the pulse wave reaches the peripheral vessels by the time of the origin (Nurdergraaf, 1961).

It thus follows from the above that the origin of ballistic forces is conditioned not so much by the mechanical processes of myocardial contraction as by the blood flow, that is by the hemodynamic factor. Such an opinion was expressed also by Dock and Grandell (1957). Jacobs (1954) stresses the importance of the state of the arteries and arterioles, and Nurdergraaf (1961) notes the role of the venous vessels. He finds that the small arteries account for 20% of the total ballistic effect. Winer (1958) believes that if the HI wave reflects only systole, then the vascular mechanisms play a certain part in the genesis of IJ and JK in addition to the cardiac factors. In this respect one cannot agree with certain authors who /242 claim that vascular factors play no part at all in the origin of ballistic forces. A. I. Hefter (1959) is right in his belief that ballistocardiography makes it possible to evaluate the role of both cardiac and extra-cardiac factors in the pathogenesis of circulatory insufficiency.

We have described the configuration of individual waves. But the principle used to determine the width and amplitude of the ballistocardiographic waves is somewhat different from the one employed in electrocardiography. Each ballistocardiographic curve is analyzed from the point of view of qualitative and quantitative indices (Fig. 88). The first step is to take a close look at the curve as a whole and to evaluate it from the point of view of technical completeness. The beginning of the systolic complex is then determined. For that purpose, a straight line is drawn from the peak of the R wave on the electrocardiogram to the ballistocardiographic curve (the electrocardiogram is recorded on the first channel of the apparatus, and the ballistocardiogram on the second). The first wave occurring beyond that line will be the H wave, as it is known that the onset of the mechanical systole is a little later than that of the electrical systole. It is then no longer difficult to designate the other waves. The iso-electric line should also be determined. This can be achieved by using the level of the last residual wave or the level of the beginning and end of the N wave of the preceding complex as a guide. We cannot agree with the proposal made by Moss (1961) that the horizontal line running along the peak of the H wave be arbitrarily adopted as an iso-electric line.

The following is an analysis of the complex according to the following factors:

1. Determine the time intervals, in seconds, between the apex of the R wave of the electrocardiogram and the peak of the corresponding ballistocardiographic wave. The following intervals can be determined in this way: R-H, R-I, R-J, R-K and R-L. We do not agree with some authors who propose that these intervals be measured from the Q wave rather than the R wave of the electrocardiogram, because from a practical point of view it is easier and better to determine the R wave than the Q wave (there can be no serious objections to this from a theoretical point of view).

2. Determine the time intervals within the ballistocardiographic complex from the apex of one wave to the apex of the following wave. The following intervals can be determined, in seconds, by this method: H-I, I-J, J-K, K-L, H-K and I-K.

3. Determine the absolute and relative amplitude of the ballistocardiographic waves. The amplitude of HI, IJ, JK and KL is measured in millimeters, and the coefficients of HI/IJ, JK/IJ, and KL/IJ are calculated. These coefficients provide a comparative evaluation in relation to the magnitude of the IJ wave, as there is a fairly clear-cut correlation between that and the other waves of a normal ballistocardiogram (Van Lingen, Lister and Elzas, 1956). Other proposals have also been made. Arbeit et al., (1957), for example, advises only the determination of the H wave amplitude and HK/HI coefficient, while Moss (1960) suggests that the HI angle and HK/HI coefficient be determined. /243

All of the mentioned time intervals and wave amplitudes are determined on a ballistocardiogram of a quietly breathing patient, and the arithmetic mean is established from the measurements of ten successive complexes. The intervals must be measured in ten complexes because (as we will explain later on) the nature of the curve changes in the different phases of the respiratory process, and the arithmetic mean provides the average magnitudes of two-thirds of the respiratory cycles. This is not a generally accepted approach. Moss (1960), for example, recommends such a determination in three successive complexes, and Starr (1958) believes that it would be sufficient to measure only the waves with the highest and lowest complexes.

4. Determine the three functional indicators: The respiratory factor is determined by the following formula $\frac{\text{maximum IJ amplitude}}{\text{minimum IJ amplitude}}$. The appropriate wave is selected from ten successive complexes recorded during conditions of quiet respiration. This indicator provides a quantitative idea of the changing wave amplitudes in various phases of respiration. The ballistocardiographic index is expressed by the following formula $\frac{\text{minimum IJ amplitude}}{\text{MSB (maximum speed of body movement)}}$.

Here again the appropriate wave is selected from ten successive complexes recorded in conditions of quiet breathing. According to Dock, Mandelbaum and Mandelbaum (1953), the maximum speed of body movement (MSB) corresponds

to the forces producing the maximum body movement in a longitudinal direction. In actual practice, it is usually the JK wave. This index gives an idea of the respiratory waves and, especially, the force of cardiac contraction.

The intrasystolic index equals the following ratio $\frac{\text{H-K interval}}{\text{R-K interval}}$ in

percentage terms, and the arithmetic mean is used in this case. The significance of this index is that the R-K interval may be considered as an equivalent of the mechanical systolic duration, the R-H interval as a reflection of the filling phase, and the H-K interval as a reflection of the expulsion phase. Consequently, the intrasystolic index points to a relatively lengthy expulsion phase in mechanical systole. It is similar in principle to the systolic index of an electrocardiogram, but the latter index defines the relative duration of the general electrical systole in one full cardiac cycle. /244

Table 7

Normal indexes of a ballistocardiogram recorded
by the R. M. Bayevskiy method (1962)

Indexes		Age groups (years)							
		15-20	21-25	26-30	31-35	36-40	41-45	46-50	Over 50
Duration of interval, in seconds	H-I	0.55	0.061	0.060	0.060	0.065	0.083	0.052	0.056
	I-J	0.083	0.084	0.086	0.085	0.083	0.083	0.077	0.078
	J-K	0.095	0.098	0.099	0.008	0.100	0.106	0.106	0.103
	K-L	0.106	0.111	0.103	0.106	0.102	0.103	0.105	0.108
	H-K	0.233	0.243	0.245	0.243	0.248	0.272	0.235	0.237
Wave amplitude, in millivolts	HI	0.72	0.82	0.69	0.65	0.66	0.58	0.42	0.39
	IJ	1.62	1.58	1.50	1.45	1.43	1.14	1.14	1.23
	JK	2.63	2.41	2.28	2.23	2.26	1.84	1.87	1.75
	KL	2.71	2.33	1.87	1.89	1.97	1.66	1.71	1.60
Coefficients	HI/IJ	0.45	0.52	0.43	0.43	0.43	0.45	0.37	0.32
	JK/IJ	1.67	1.53	1.37	1.58	1.62	1.60	1.64	1.17
	KL/IJ	1.46	1.64	1.22	1.37	1.38	1.45	1.50	1.30
	Ballist. index	0.34	0.39	0.36	0.37	0.33	0.38	0.42	0.40

5. To determine the configuration of the waves, their changes and the degree of pathology (see above).

A number of studies citing the magnitude of these waves, intervals and indexes within the norm are found in literature (S. S. Belousov, 1958; A. I. Hefter and colleagues, 1958; S. M. Gusman and E. Sh. Halfen, 1958; etc.). Table 7 shows the indexes of a ballistocardiogram recorded by the R. M. Bayevskiy method (1962). The standards we have developed are shown on pp.

363 and 364. It should be pointed out that since the apparatuses are not based on a standard design, different standards should be established for the different tables and data units.

Different physiological conditions produce certain changes in the picture of a normal ballistocardiogram. The constitutional characteristics of the subject and the sex do not materially affect the formation of the curve. The weight and size of the test subject cannot produce a substantial effect in direct-method ballistocardiography, except when they are considerably greater than the average magnitudes or the given age and sex. We consider it necessary to dwell only on the effects of respiration and age.

IV. THE BALLISTOCARDIOGRAM AND RESPIRATION

The original researchers in the field of ballistocardiography noted that /245 in a healthy person the IJ wave amplitude increased during inspiration and decreased during expiration (Fig. 90). The mechanism of this phenomenon can be understood if the diphasic phenomena occurring during respiration is understood, and that it can affect the activity of the heart. In the first place, this is an anatomical factor. It is a known fact that the position of the heart changes in the act of respiration. During inspiration the long axis of the heart assumes a vertical position, and during expiration its position is more horizontal; that movement plays a very insignificant part. Starr's observations (1940) of artificial respiration under positive pressure show that although this process produces similar changes in the position of the heart, the ballistocardiogram reveals a reverse pattern. If the mechanical factor actually produced a great effect, an increase of all the H waves would be expected during expiration as the heart assumes a horizontal position, and would be accompanied by an amplification of the vertical component of the apical impulse which plays some part in the formation /246 of the H wave. However, as Brown et al. (1952) points out, such a phenomenon is observable in only 55% of the test subjects. Finally, it should be borne in mind that the changes of the vertical and lateral ballistic forces in accordance with the respiratory phases are on a small scale, and cannot be responsible for the fairly large changes in the amplitude of the ballistocardiographic waves observable in the different phases of respiration. R. M. Bayevskiy and V. K. Sel'tser (1960) find that the respiratory changes on the ballistocardiogram are also due to the changing density of the mediastinal tissues and, therefore, the respiratory fluctuation of the forces transmitted from the heart to the body.

Second, is the hemodynamic factor. It is known that intra and extra cardiac hemodynamics, especially the hemodynamics of the lesser circulation undergo a change during the respiration. It follows from the above-mentioned observations by Starr that the intrapleural pressure plays an important part. A change in its normal dynamics (it is known that the intrapleural pressure decreases during the normal inspiration, but increases during artificial inspiration under positive pressure) produces a corresponding change in the respiration pattern on the ballistocardiogram. In their experiments involving cardiometric measurements and the use of motion pictures, Cahoon,

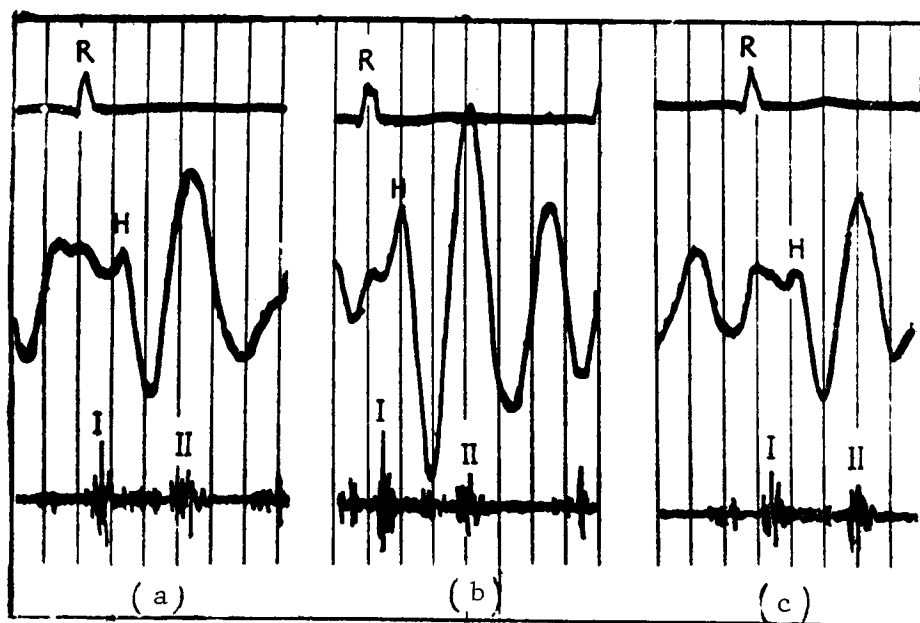


Fig. 90. The effect of respiration on the ballistocardiogram. A ballistocardiogram of patient A. A., age 30, with the following diagnosis: mitral stenosis. (a) When breathing quietly; (b) wave amplitude increases by delayed inspiration; (c) wave amplitude decreases by delayed expiration (the time divisions in all the figures amounts to 0.1 sec.)

Michael and Johnson showed that inspiration decreases the diastolic dimensions and stroke volume of the ventricles as well as the volume of the left atrium, and increases the volume of the right atrium. The authors assumed that inspiration was accompanied by an incomplete filling of both ventricles. The original experiments carried out by Boyd and Patras in the same year (1941), involving a cardiometric recording of a closed thorax, led them to different conclusions. They proved that if the ventricles are under constant atmospheric pressure, the diastolic volume and stroke volume of the heart are actually considerably decreased during inspiration. However, the measurements made under intrathoracic pressure, which are normal to ventricular activity, actually reveal an inspiratory increase in the diastolic as well as stroke volume of both ventricles. This pattern was confirmed also by the experiments carried out by Patras, Brookhart and Boyd (1944). They observed an inspiratory acceleration and expiratory deceleration of the filling process in conditions of a lengthened diastole produced by excitation of the vagus nerve.

It follows from the above-mentioned data that the increase in the ballistic forces during inspiration is responsible for the increase in the stroke volume of the heart. But this brings up a considerable contradiction. If the heart expels more blood during the inspiration, how is the reduction of the systemic arterial pressure possible in this same phase of the respiratory cycle? This seeming contradiction was explained in the experiments carried out by Shuler et al. (1942) who made a motion-picture recording of

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the cardiac contractions through an opening in the thorax, and proved that the inspiration increases the diastolic and stroke volume of the right ventricle, and decreases them in the left ventricle; the situation is reversed during the expiration. That means that not all of the large quantity of blood coming into the lesser circulation during the inspiration flows into the left ventricle. Consequently, the reduction of the systemic arterial pressure during the inspiration is due to the reduction of the stroke volume of the left ventricle. Moreover, the respiratory changes in the general stroke volume occur simultaneously with the hemodynamic changes in the right ventricle.

These experimental observations were eventually confirmed in clinical practice. Using the method of cardiac catheterization, Lauson, Bloomfield and Cournand (1946) showed that in a human being the aortic pressure and the stroke volume of the left ventricle is reduced by 5-15% during inspiration, while the pressure in the pulmonary artery and the stroke volume of the right ventricle are increased by 15-40%.

Experimental and clinical data justify the conclusions that the respiratory changes in the amplitude of the IJ wave are connected with the changes in the general stroke volume of the heart. The inspiratory increase in the ballistic forces are associated primarily with the activity of the right ventricle, and that the right ventricle plays an important part in the over-all formation of the ballistocardiographic curve. Brown and de Lalla (1950) showed that the inspiratory increase in the ballistic forces occurs in parallel with and in direct proportion to the increase of the systolic volume in the right ventricle and its decrease in the left ventricle. Dock, Mandelbaum and Mandelbaum (1953) found that the factors associated with the activity of the right ventricle amount to 80% of the total magnitude of the ballistic impulse which accounts for the formation of the IJ wave, although the latest information produced by R. M. Bayevskiy and V. K. Sel'tser (1960) stresses the more important role of the left ventricle in the formation of the ballistic forces. The tentative assumption may be made that during the inspiration the IJ wave reflects the function of the right heart and during the expiration the performance of the left heart. (G. A. Vitenshteynas, 1958; A. N. Kokosov, 1958). It should be pointed out, however, that ballistic forces are generated in both the greater and lesser circulations but, as Nurdergraaf (1961) also points out, the blood flow in the greater circulation is naturally more important. Moreover, the predominance of right ventricular ballistic forces is also due to the fact that the rate of the blood flow from the right ventricle is higher than that from 248 the left ventricle, and there is a direct ratio between the rate of speed and the magnitude of the ballistic forces.

What is the explanation for such an "original" physiological pattern of activity of the two halves of the heart in different phases of the respiratory act? It is a known fact, that the intra-thoracic pressure is reduced during inspiration. The result of this phenomenon is an intensified venous inflow into the right atrium and an increased stroke volume of the right ventricle. This is facilitated also by such factors as the compression of the liver by the diaphragm, the expansion of the pleural cavity during in-

spiration and particularly the increase in intra-abdominal pressure which, in turn, intensifies the venous inflow into the right atrium due to the increasing gradient between the right atrium and the vena cava. The stroke volume of the left ventricle is reduced, but theoretically should be increased following the increase in the volume of the right ventricle. Such a reduction of the left ventricular volume cannot be explained by the changing intra-thoracic pressure, as the "pulmonary veins - left atrium" system is under over-all intra-thoracic pressure. The change in the intra-thoracic pressure cannot change the usual pressure gradients which determine the normal blood flow in the system. The explanation for that physiological mechanism is that the lungs expand during the inspiration distending the walls of the bronchi, pulmonary capillaries and venous vessels, and the result is a temporary delay of the blood in the lesser circulation. This phenomenon results in a reduced inflow into the left heart.

The reverse picture is observable during expiration. The increase in intra-thoracic pressure leads to an increasing flow into the left atrium while the venous inflow into the right atrium is reduced as the pressure gradient between the right atrium and the vena cava is lowered.

The above described relationship between the pulmonary circulation and respiration does not justify an over-importance of the respiratory movements in the hemodynamics of the lesser circulation. V. V. Parin and F. Z. Meyerson (1960) justly criticize this tendency, and point to the importance of the nervous regulation in the system of pulmonary circulation.

It will be seen later that physiological and especially pathological conditions play an important part in the changes produced on the ballistocardiogram by breathing.

V. PATHOLOGICAL CHANGES ON THE BALLISTOCARDIOGRAM

Changes in the wave configuration and amplitude, an increase or decrease in the time intervals and respiratory fluctuations may be observed in pathological conditions. The disruption of the normal synchronism between the left and right halves of the heart, in addition to the disruption of the contractile capacity of the myocardium, plays an important part in the origin of these changes. /249

An increase in the H wave amplitude assumes considerable pathological importance. Dock, Mandelbaum and Mandelbaum (1953) associate the origin of the increased H wave, especially its early origin, with the functions of the atria. And they attribute it to the reverse blood flow which occurs after the ventricle has been rapidly filled with blood. This explains also the high H waves occasionally observable in cases of atrial flutter, even though in this case the H wave frequently has a reduced amplitude and is split (Fig. 96) or missing. A considerable increase in the H wave amplitude produces a so-called early M-shaped complex (Starr and Wood, 1943) as the H wave amplitude is almost equal to the J wave amplitude (Fig. 91); such complexes are very important in the clinical evaluation of a ballistocardiogram

(Z. I. Yanushkevichus and G. A. Vitenshteynas, 1959). A splitting of the H wave is occasionally observable. The bifurcation of the H wave in the case of mitral stenosis is due to the nonsynchronous movement of the atrio-ventricular septum and the blood in the right and left auricles, as the tricuspid valve is pliable, but the bicuspid valve, on the other hand, is rigid (Dock, Mandelbaum and Mandelbaum, 1953) (Fig. 92).

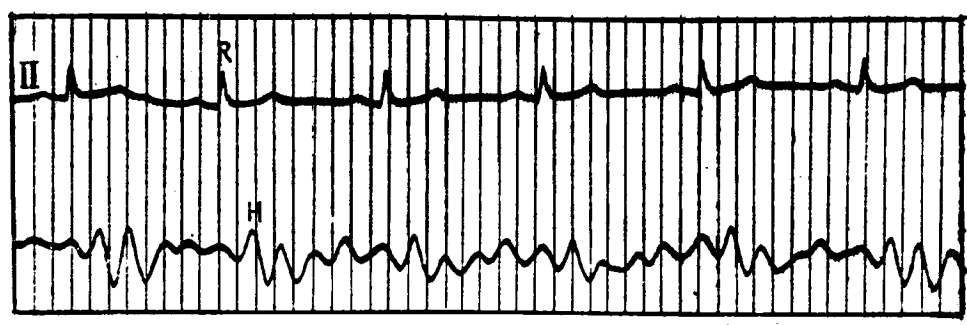


Fig. 91. Pathological changes on the ballistocardiogram. A ballistocardiogram of test subject K. P., age 54, and essentially healthy. Reduction of J wave amplitude, and increase of H wave amplitude: early M-shaped complex due to H.

Starr (1958) attributes a great deal of importance to the form of the I wave, and believes that its depth as well as the incline of the HI wave are indicative of an initial acceleration of the blood flow which may be designated as a "click" of the cardiac contraction. The same opinion is held by Moss (1960) who finds that the incline of the HI wave reflects the speed of blood flow. Mena (1955) finds that the HI changes point to a disrupted process of blood flow from the ventricles. In experimental conditions, if the synchronous inflow into the aorta and pulmonary artery is characterized by a very low initial acceleration, the I wave is not detected. It may be said that this wave loses its normal form very early in the gradual weakening of cardiac contractile force. An increase in the I wave (Fig. 92) is frequently observable in pathological conditions. It is increased in cases of coarctation of the aorta. When the I wave is split, it may be difficult to differentiate it from the H wave. /250

The amplitude of the J wave may increase or decrease (Fig. 91 and 94). Starr (1957) cites a long list of diseases in which hyperkinemia or hypokinemia (an increase or decrease of the ballistocardiogram amplitude) is observed. According to him, hyperkinemia is found in healthy people, in cases of hyperthyreosis without cardiac complications, aortic regurgitation, severe exhaustion, patent ductus arteriosus, peripheral arteriovenous aneurysms, anemia, and chronic pulmonary diseases. Hypokinemia is found in moribund patients, in hemorrhagic shock, cardiac insufficiency, organic heart disease without decompensation, coronary disease (coronary occlusion immediately followed by the development of an infarction) hypertension, endocrine diseases (myxedema, hypophysis and adrenal gland diseases). The splitting of the J wave (Fig. 92) resulting in the formation of a late M-

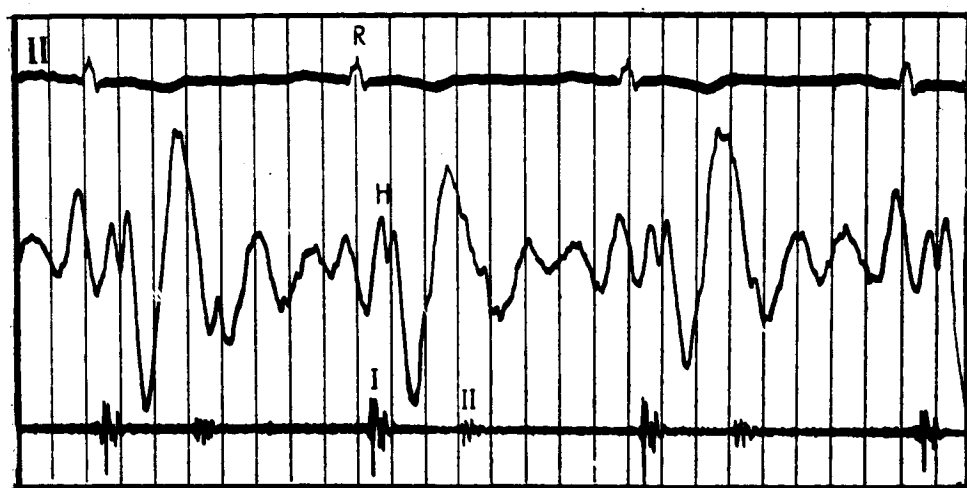


Fig. 92. Pathological changes on the ballistocardiogram. A ballistocardiogram of patient M. A., age 41, with a diagnosis of mitral stenosis. Split H wave, deep I wave and periodic J wave splitting (on the third channel is a phonocardiogram).

shaped complex is frequently observable in pathology (Starr and Wood, 1943). The J wave is split by the nonsynchronous contraction of the two ventricles. If the split is intensified during inspiration and one of the apices is increased, the process is produced by the lagging right ventricular forces. If a similar picture is observable during the expiration, the lagging left ventricular ballistic forces may have something to do with it. Bodrogi (1958) finds that the late M-shaped complexes may be observed in cases of a tetralogy of Fallot, i-v septal defects, and stenosis of the pulmonary artery. Such complexes are found also in myocardial infarcts.

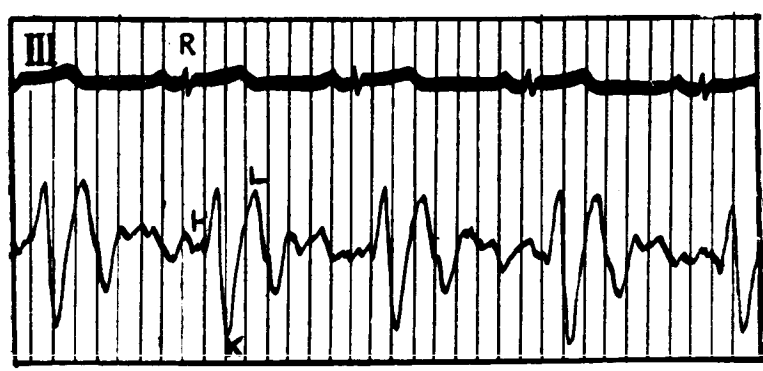


Fig. 93. Pathological changes on the ballistocardiogram. A ballistocardiogram of patient B. T., age 37, with following diagnosis: hypertension, IIb degree. Deep K wave, late M-shaped complex produced by pronounced L wave, and R-H interval somewhat increased.

Hypertension, according to a number of authors, increases the K wave

(Fig. 93). But we have frequently observed a decrease and even an amputation of the K wave in cases of hypertension. A characteristic increase in the amplitude or absence of the K wave is observable in coarctation of the aorta. (Nickerson et al, 1950). It is decreased also in cases of cardiac insufficiency. A split K wave (Fig. 94) is frequently observed in pathology. Braunstein (1953) calls attention to the wide K waves which Starr and Wood (1943) refer to as late downstroke types. He finds that the contraction of a healthy heart is accompanied by a "click", and that a large quantity of blood flows into the aorta and pulmonary artery early in the systolic period, whereas in a defective heart, the weak contractions expel the blood into the vessels slowly, and the maximum expulsion occurs in the late systolic period. As a result of such a mechanism, the K wave acquires the form of a late downstroke.

The high L waves observable in mitral stenosis with insufficiency, in coronary insufficiency, and other diseases may be due to the fact that during the isometric relaxation of the ventricles the atrioventricular septum moves up and the influx of blood to the atria is slowed. (Dock, Mandelbaum and Mandelbaum, 1953). If high L waves are present, we refer to such complexes as late M-shaped complexes produced by the L wave (Fig. 93). /252

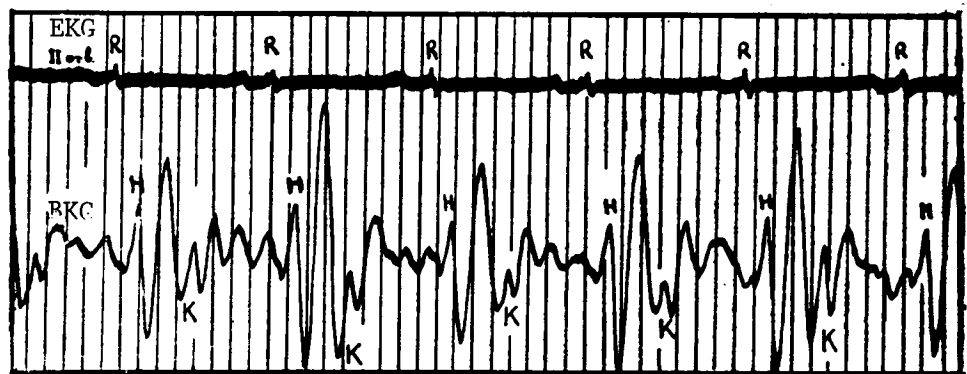


Fig. 94. Pathological changes on the ballistocardiogram. A ballistocardiogram of patient M. L., age 69, with following diagnosis: hypertension, stage IIIb, and atherosclerotic heart disease. Increased systolic wave amplitude, the K wave is split and the R-H interval increased (0.13 sec.).

The pathological changes of the M and N waves are still practically unknown. The post systolic waves occasionally resemble a systolic wave silhouette which is known as the "echo phenomenon" (Fig. 95). Observations and experiments involving the reproduction of pathological complexes have prompted Starr (1958) to conclude that when low systolic waves are present the pronounced diastolic complexes are indicative of a relaxation of the myocardial contractile function. The changes of the L, M and N waves are, on the whole, associated with the disrupted hyperemic period of the ventricles.

In pathological conditions, changes are observable also in the time intervals. Hypertension and atherosclerosis frequently reveal an increase in the R-H interval (Lin'-Chen, 1957 and 1958; Ye. V. Erina, 1960). In our observations, this phenomenon (Figs. 94 and 95) appears to be conforming to consistent rules, and the R-H interval in hypertensive patients increased in keeping with the increasing gravity of the disease. We have observed such an increase also in rheumatism (Z. L. Dolabchyan and M. A. Yesayan, 1961), under the effect of chloroprene (Ye. I. Gasparyan and Z. L. Dolabchyan, 1963) as well as in all cases involving a disruption of the myocardial contractile function due to dystrophic or sclerotic changes in it. Certain cases of hypertension also involve an increase of the H-K interval, but there the intra-systolic indicator does not increase as the elongation of the entire mechanical systole (R-K) is primarily due to the increasing stress phase (R-H). Lin'-Chen and Ye. V. Erina rightly consider the decreasing I-K interval as a manifestation of aortic stenosis.

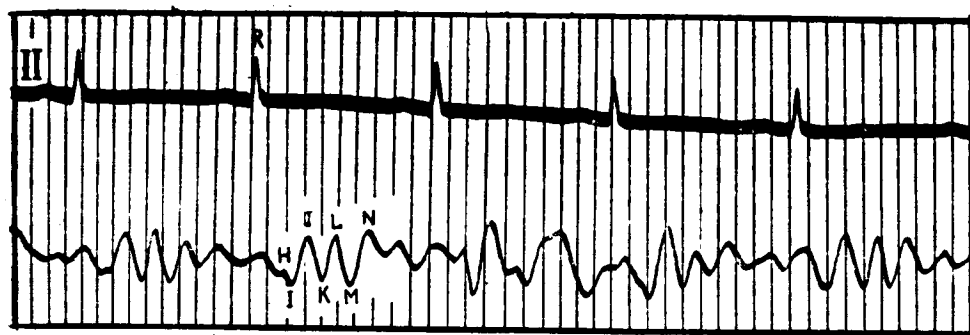


Fig. 95. Pathological changes on the ballistocardiogram. A ballistocardiogram of patient, Zh. P., age 52, with following diagnosis: hypertension, stage IIb. Reduced systolic wave amplitude; the diastolic waves are increased and they repeat the systolic wave amplitudes, the echo phenomenon. The R-H interval = 0.18 sec. /253

It should be pointed out that in the initial period of the development of pathological changes in the myocardium, the changed ballistocardiographic complexes, particularly the systolic ones, are observable mostly during expiration. It is difficult to explain this phenomenon. In all probability, there is some mechanism at work here, such as the expiratory reduction of the cardiac ballistic forces resulting from a diminished inflow of blood into the right atrium. The importance of the extent of ventricular filling in shaping the systolic complexes is indicated by the fact that in cases of atrial flutter the shape of the complexes changes according to the duration of the diastole of the preceding cardiac cycle. Another indication of it is/254 that if an extrasystole develops against the background of a pathological picture, the complex frequently assumes a more or less normal appearance after a compensatory pause. A further development of pathological changes in the myocardium reveals changed ballistocardiographic complexes also during inspiration.

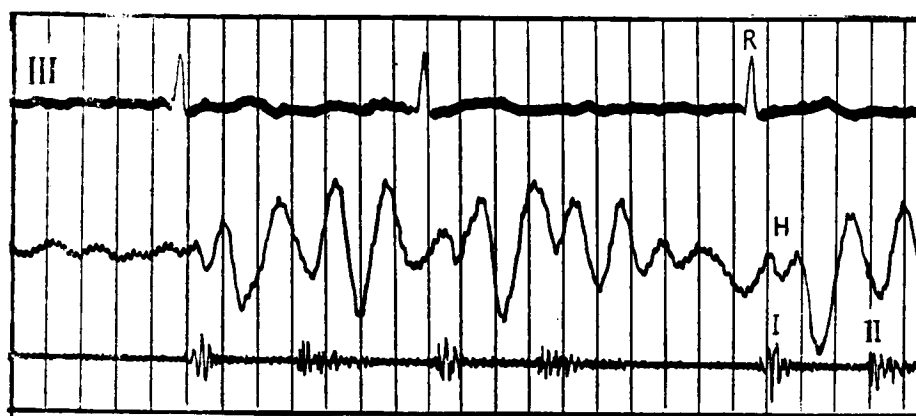


Fig. 96. A ballistocardiogram showing atrial flutter. A ballistocardiogram of patient A. T., age 20, with following diagnosis: mitral stenosis. In the case of an atrial flutter, the H wave remains, becomes split, and the form of the systolic complexes depends on the duration of the diastole of the preceding cycle.

We pointed out above that pathological conditions change the picture of normal respiration. This information is discussed in the following section.

VI. CLASSIFYING THE DEGREES OF PATHOLOGY ON THE BALLISTOCARDIOGRAM

Brown and de Lalla (1950) suggest that the RVI (respiratory variation index), which is expressed by the following formula, be calculated in order to make a quantitative determination of the respiratory variation on the ballistocardiogram:

$$RVI = \frac{(\text{maximum minute volume during inspiration}) - (\text{maximum minute volume during expiration})}{\text{body surface}}$$

The normal range of the index fluctuates from 0 to 450 cm³ a minute per M² of body surface. The authors define the minute volume according to the Starr formula (see page 260). In actual practice, however, the minute volume of the heart may be left undetermined as a comparison of the IJ wave amplitudes in various phases of respiration will provide an idea of that index.

Basing their argument on that possibility, Brown et al (1952) proposed the following classification of pathological ballistocardiograms.

Zero degree (Fig. 97). A normal curve with usual respiratory variations.

First degree (Fig. 98). The waves are regularly and easily detected, /255

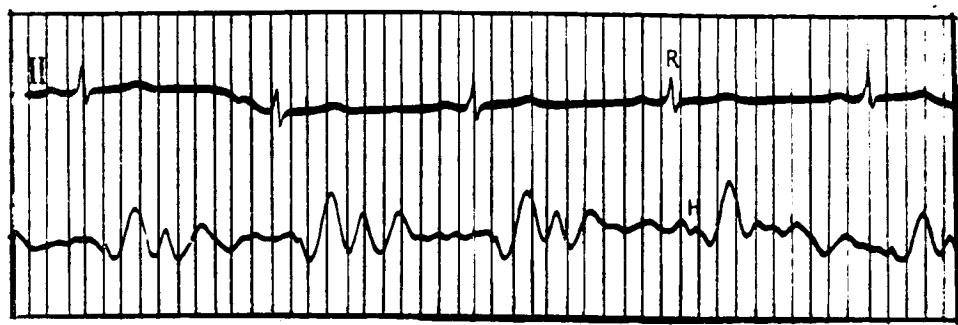


Fig. 97. Degrees of ballistocardiographic deflections. A ballistocardiogram of patient S. S., age 38, with following diagnosis: hypertension, stage IIb. The zero degree curve is based on the Brown method (explanation found in text).

the IJ wave has a normal amplitude during inspiration but is reduced during expiration. The respiratory variation index is increased. Characteristic of this degree is that during expiration the individual IJ waves are decreased by more than one-half amplitude of the maximum IJ wave during inspiration.

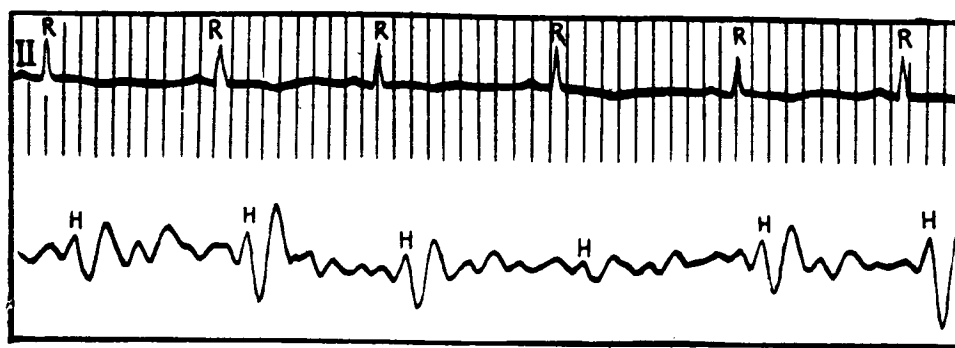


Fig. 98. Degrees of ballistocardiographic deflections. A ballistocardiogram of patient S. A., age 25, with following diagnosis: hypertension, stage IIa. The first degree curve is based on the Brown method (explanation found in text).

Second degree (Fig. 99). The amplitude of the IJ wave is reduced in more than half of all the complexes, and a similar reduction is observable also during inspiration.

Third degree (Fig. 100). The amplitude of all the complexes is reduced, their regularity and clearness is adversely affected and the complexes are slightly differentiated only during inspiration.

Fourth degree (Fig. 101). There is a sharp decrease in the amplitude of all the complexes, and they are not differentiated. Irregular variations are observable.

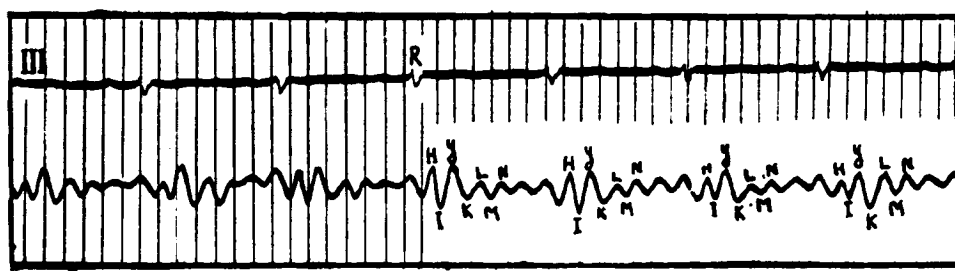


Fig. 99. Degrees of ballistocardiographic deflections. A ballistocardiogram of patient M. Kh., age 47, with following diagnosis: systemic scleroderma. Second degree curve is based on the Brown method (explanation found in text).

A similar classification is proposed by Dock, Mandelbaum and Mandelbaum ^{/256} (1953) and V. V. Parin (1956). Using the major three types of pathological complexes described by Starr and Wood (1943) as a basis (the early and late M-shaped complexes and the late downstroke type complexes), Wade, Fulton and McKinnon (1956) propose the following three degrees of pathology on the ballistocardiogram.

First degree. The pathological complexes are detected only during expiration.

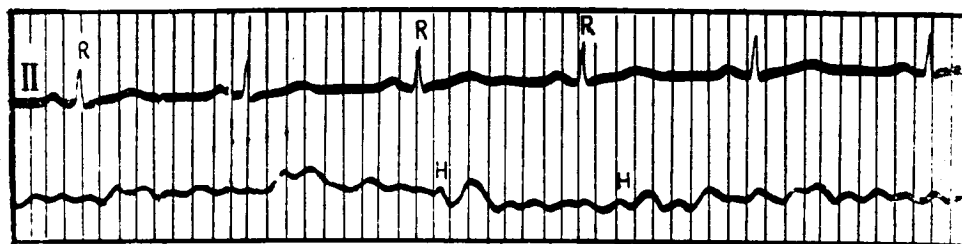


Fig. 100. Degrees of ballistocardiographic deflections. A ballistocardiogram of patient P. A., age 68, with following diagnosis: atherosclerotic heart disease and hypertension, stage Ib. Third degree curve is based on the Brown method (explanation found in text).

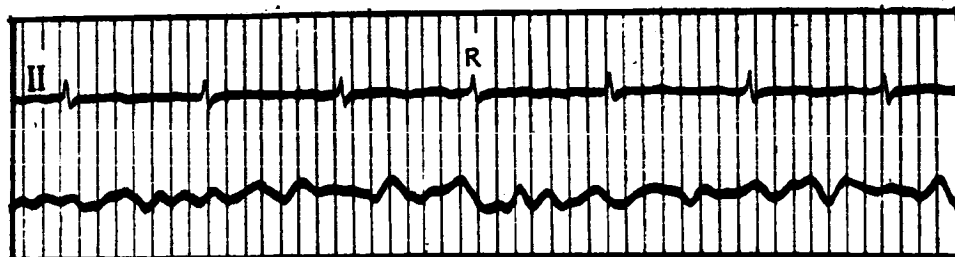


Fig. 101. Degrees of ballistocardiographic deflections. A ballistocardiogram of patient A. G., age 60, with following diagnosis: atherosclerotic heart disease (infarction in anamnesis), and hypertension, stage Ib. Fourth degree curve is based on the Brown method (the single complexes on the given segment are somewhat differentiated).

Second degree. All complexes are pathological, but they are repeated and can be differentiated.

Third degree. Illegible complexes; they can be differentiated only by means of an electrocardiogram.

According to the A. L. Limcher (1959) classification, the degrees of deflection are determined by the changes occurring during quiet respiration and during suppressed respiration: in the first degree the pathological changes occur only in the expiration phase of quiet respiration, in the second degree most of the complexes change during quiet respiration but become normal when respiration is suppressed (frequently during inspiration), and in the third degree of deflections all the complexes are changed and become normal when respiration is suppressed. /257

Attaching much pathological significance to the increase in the H and L waves, G. A. Vitsenshteynas (1958) suggests that the names of these waves be added to the Brown-type curve representing the respective degrees of pathology. For example, if a second degree pathological ballistocardiogram reveals high H waves, it should be designated as "pathological curve II-H degree", and if high L waves are observable, II-L degree should be indicated. N. V. Zhuravleva (1959) finds that the changes in the magnitudes and forms of the individual waves occur just as frequently as the "respiratory" changes in the amplitude of the systolic complexes. She therefore proposes four groups of ballistocardiographic changes which she designates as insignificant, moderate, pronounced and sharply pronounced. A more complicated classification is proposed by Moss (1961): it is based on seven quantitative measurements and six descriptive concepts.

We use the Brown classification to which Lin'-Chen's (1957) modifications are frequently added. That author distinguishes three degrees of pathological changes, insignificant, average and significant changes, each degree being divided into subdegrees a and b. The Ia degree corresponds to the I degree of the Brown classification; observable in the Ib degree are individual or split IJK complexes. In the IIa degree, the changes or splitting of the IJK complexes are observable in less than half of all the systolic complexes; the IIb degree corresponds to the II degree of the Brown classification. The III degree corresponds to the changes of the III and IV degrees of the Brown classification. Thus Lin'-Chen distinguishes six degrees of ballistocardiographic deflections from the norm which make it possible to detect the more subtle changes.

What phenomena is the Brown classification based on? It is known that the normal respiratory variation index is determined by the dynamic mutual relation between the functions of the right and left ventricles. If the left ventricle is injured, this index increases as the influence of the right ventricle become predominant. Moreover, the reduction or even disappearance of the left ventricular ballistic forces leads to a still greater reduction in the amplitude of the complexes during expiration. The isolation of the injury to the right ventricle will reduce the index as the ballistic impulses of the left ventricle will thus become prevalent.

It should be borne in mind, however, that in actual practice both ventricles are frequently affected simultaneously, and in numerous cases the left ventricle is affected first and is followed by a disturbance of the activity of the right ventricle. It is a known fact that in many cases an injury to the left ventricle results in the development of hypertension in the pulmonary circulation due to the rising pressure in the left atrium and the operation of the F. Ya. Kitayev reflex. This phenomenon (which was described in 1931) is that the rising pressure in the left atrium and pulmonary veins results in a reflex from the walls of these areas which produces a vasoconstricting effect, a contraction of the pulmonary arterioles. In the later periods, the pulmonary hypertension is maintained by the organic changes in the vessels of the lungs. The significance of pulmonary hypertension can be easily seen if we bear in mind that the scale of the changes may reach a magnitude never observed in systemic hypertension (V. V. Parin and F. Z. Meyerson, 1960). In addition to such hemodynamic factors whereby the right ventricle is affected by the disruption of the activity of the left ventricle, there is also the anatomical factor which is responsible for the extension of the pathological process from the left ventricle to the neighboring sections of the right ventricle.

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In all these mechanisms, de Lalla and Brown (1950) attach great importance to the retention of blood in the lungs which acts as a cushion between the right and left ventricles. They find that in certain conditions the increasing respiratory variations are probably connected with the reduction of that retention as a result of increasing intrapulmonary pressure during the expiration or a longer retention of the blood in the peripheral and celiac vessels.

Thus, it may be said that the first degree of deflections reflects only an insignificant disruption of the contractive capacity of the myocardium. It is frequently connected with extra cardiac factors and may be determined by definite disruption of cardiac activity, particularly the activity of the left ventricle. The second degree deflections reveal a moderate disruption of cardiac activity even if it is still not very pronounced clinically. A reduction in the wave amplitude and increase in the R-H interval point to a weakening of the contractile function of the myocardium, while a wave deformation is indicative of a disrupted normal synchronism between the two halves of the heart. The third degree indicates a considerable disruption of cardiac activity. The sharp reduction in the amplitude of the waves, their differentiation only during inspiration and considerable deformation of the waves and changes of the time intervals, particularly R-H, R-K and H-K, are indicative of considerable weakening of the contractile capacity of the myocardium and asynchronism between the left and right ventricles. The fourth degree indicates a very pronounced disruption of cardiac activity and circulation as a whole.

VII. ARTIFACTS IN THE BALLISTOCARDIOGRAM

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We discuss this problem separately in view of the fact that artifacts frequently produce great difficulties and may become the source of serious

mistakes. Starr (1958) formulates the following law of artifacts: "Whatever is not regularly repeated does not deserve any attention". It should be remembered here that the observation of recurrence should be made between complexes which occupy similar places in the respiratory cycle. According to Starr (1958), the artifacts are divisible into five categories.

Respiratory artifacts. These artifacts are frequently observed in the form of cyclical (according to the respiratory phases) fluctuations of the isoelectric line. They are intensified with increasing respiratory frequency, and the contractions of the diaphragm produces new artifacts which are easily detected. This can be done by tracing the respiratory act. These artifacts are particularly pronounced when the patient's breathing is irregular.

Artifacts produced by the test subject's movements. These are easily detected by their undetermined configuration and lack of recurrence. No definite pattern is noted in the appearance of these fluctuations. Artifacts produced by muscular tension change the entire background of the curve and frequently make its reading considerably difficult.

Artifacts resulting from movements inside a building. These produce small fluctuations characterized by a sudden beginning and end and a relatively high frequency. They are caused by moving elevators, vehicles on the street, etc.

Artifacts produced by interference. Interference occurs whenever a preceding phenomenon is superimposed on the following one preventing its formation. A ballistocardiogram should not be recorded in cases of pronounced tachycardia, as the post systolic waves are superimposed on the systolic ones as a result of interference and considerably distort the curve.

Resonance-produced artifacts. The resonance phenomenon occurs when the ratio between the frequency of the ballistic force and the natural body frequency is 1. In this case the amplitude of the following waves increases and the normal picture is distorted. This phenomenon is possible even when the rhythm of cardiac activity is normal, especially if the recording is made by the direct method, when the body movements continue for some time after the effect of the ballistic force. The K and L wave amplitude usually increases in such cases.

The artifacts connected with the inaccuracies in the recording apparatus may be added to the artifacts mentioned above. This is particularly important when the Dock direct method is used.

As pointed out above, the artifacts are primarily due to the failure to observe the techniques and methods of ballistocardiography. They do not occur when the necessary rules are observed, and do not deteriorate the technical quality of the recorded curve. /260

M. N. Tumanovskiy and Yu. T. Safonov (1960) describe a simple method of double mechanical damping which helps eliminate extraneous mechanical vibra-

tions, muscular tremor and inertial body oscillations. Tightly packed sand-bags are placed under the table legs, the test subject is placed on a soft cotton mattress extending down to his knee joints, which rest on a sand elevation equaling the thickness of the mattress in the area of the Achilles tendons.

VIII. THE BALLISTOCARDIOGRAM AND CARDIAC STROKE VOLUME

The characteristic feature of the initial period of development of ballistocardiography was that all the researchers saw in it a simple method for the quantitative determination of the cardiac stroke volume. A number of formulas were proposed, all of them based on the proposition that the wave amplitude of the systolic complex is proportional to the cardiac stroke volume. Henderson proposed such a formula for determining the cardiac stroke volume back in 1905. Another formula was developed by Abramson in 1933. A great deal of importance was attached to the formula submitted by Starr (et al) in 1939. Starr and his associates began by making empirical observations during which they established the connection between the ballistic forces and the cardiac stroke volume. This information was then used as a basis for the following formula:

$$S_v = K \sqrt{(3I + 2J) A C^{\frac{3}{2}}}.$$

S_v = average cardiac stroke volume;

K = constant of 7, and sensitivity of the apparatus is such that a force of 280 grams produces a 10 mm fluctuation;

I = I wave amplitude in mm;

J = J wave amplitude in mm;

A = Cross section area of aorta in cm^2 , calculated by the following formula developed by Bazett and his colleagues (1935):

$$A = aS' + b,$$

in which S corresponds to the test subject's body surface, in M^2 , and the letters a and b represent the constant magnitudes for the appropriate age (they can be obtained from the previously compiled tables).

On checking the formula, Starr (1938) discovered a great truth: a comparison of the stroke volume, as defined by the given formula, with the data /261 produced by the ethyl-iodide method* exhibited very satisfactory results in 28 of the 30 test subjects.

Translator's note: *Fick principle using foreign gases, i.e.; Ethyl-iodide, nitrous oxide and especially acetylene.

Starr and Schroeder (1940) proposed still another formula which they had applied to 200 healthy persons (ranging in age from 20 to 84 years) to determine the normal quantities of the stroke volume. The areas, not the amplitudes, of the I and J waves are used in this formula, thus the constant has another magnitude.

$$S_v = 33 \sqrt{(2 \int I dt + \int J dt) A \sqrt{C}}.$$

These two Starr formulas produce almost the same data. Tanner (1949) eventually discovered that the area of the aorta may not be included in the formula, as in this case the constant would have a magnitude equal to 100.

But while the Starr formula, when applied to healthy people, produces correct data which approximately coincides with those obtained from cardiac catheterization (Cournand, Ranges and Riley, 1942; Tanner, 1949), the conditions in the face of pathology, is entirely different. Starr himself admitted that, and eventually came to the conclusion that ballistocardiography defines the force of the cardiac contraction rather than its stroke volume, and it is therefore difficult to find a mutual connection between the ballistocardiographic data and the cardiac stroke volume.

In 1947, Nickerson, Warren and Brannon used the formula to determine the stroke volume by means of a low frequency critically damped ballistocardiograph designed by Nickerson and Curtis (1944), and came up with the following formula:

$$S_v = \frac{5.02 F X P}{T X L} \text{ ml.}$$

F - the highest fluctuation force which is determined by the standard deflection from the effect of a 35 gram force on a table, and by measuring the distance between the peak of the first maximum downward fluctuation (I wave) and the peak of the first maximum upward fluctuation (J wave),

P - the square root of the arithmetic mean value of the systolic and diastolic pressures (within all ranges of blood pressure except local blood pressure),

T - the time interval in seconds between the I and J waves,

L - The height of the test subject in cm,

5.02 - the constant arrived at empirically by comparing the data of a ballistocardiographic examination with those obtained from cardiac catheterization. /262

Nickerson, Warren and Brannon (1947) used the given formula to get good results also in the case of patients. These results were proved correct by simultaneous determinations of the cardiac stroke volume by the catheterization of the right ventricle. Nickerson (1940) suggested additional formulas whose data also agree with the figures determined by the Fick direct method.

Introducing definite changes into the above-cited Nickerson formula and using a low frequency and partially damped ballistocardiograph and an N. N. Savitskiy (1956) mechanocardiograph, R. I. Gismatulin and M. S. Kushakovskiy (1958) have found data comparable to those obtained by plethysmography or acetylene method (Fick principle).

We do not consider it necessary to go into these problems any further, as these formulas are connected with indirect methods which we seldom use, besides, they cannot produce the precise data to meet the requirements of modern science. There is little reference at present in the ballistocardiograms to this quantitative aspect. Arnould (1951) notes that the use of these formulas must take into account as many individual characteristics of the test subject as it is practically possible to do. Even if they produce information about healthy people, these formulas lose all their value when applied to ill persons, even though Nickerson (1958) is optimistic and finds that his formula, when properly applied with certain modifications, can produce satisfactory results also in the case of illness.

IX. THE BALLISTOCARDIOGRAM AND AGE

If a ballistocardiogram of healthy young people presents a constant picture and does not reveal any pathological changes, the same ballistocardiogram of older individuals, particularly of advanced middle age and in good health does show pathological changes (Fig. 102). Moss (1961) studied the changes on the ballistocardiograms of healthy persons between 18 and 54 years of age. The initial symptoms of cardiovascular aging (first degree changes) were observed by the age of 35 in 16% of the test subjects. In advanced middle age, particularly in the fifth decade of life, there is an increase in the frequency and extent of observable pathological changes. According to S. M. Gusman and E. Sh. Khalfen (1958), healthy persons up to the age of 40 do not show any pathological changes on the ballistocardiogram. Individual pathological changes appear in advanced middle age, their number and extent increasing with age. Similar phenomena were discovered by Starr and Hildreth (1952) in repeated ballistocardiographic examinations of 80 healthy persons 10-14 years after the first examination. Starr refers to the age-connected changes on the ballistocardiogram which basically amount to a reduction of the wave amplitude, particularly a reduction of the I and J wave amplitudes, as presbycardia. It should be pointed out that age-connected changes can be found by both the indirect (Scarborough, et al. 1953; Taylor, Maloney and Keys, 1954) and the direct (Dock, 1951; Brotmacher, 1956; Z. L. Dolabchyan, 1962) recording methods. /263

We agree with Moss (1961) that age-connected changes on the ballistocardiogram should be explained by a number of factors. For example, the frequent irregularity of rhythm of cardiac activity with age is of definite though very minor importance. Some importance is attached to the observable increase in weight, as it is known that the amplitude of the ballistocardiographic waves is inversely proportional to the total weight of the table. Another factor worthy of attention is the age-connected change of the turgor

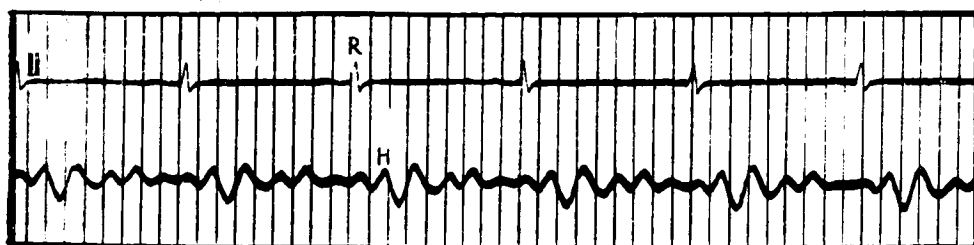


Fig. 102. Ballistocardiogram and age. A ballistocardiogram of test subject P. K., age 59, who is healthy. There is a considerable reduction in the J wave amplitude, the R-H interval is increased, the respiratory variation is not pronounced, and the second degree curve is based on the Brown method.

and elasticity of the tissues which has an important effect, especially in direct-method recording. Moss (1961) points to the age-connected change in the direction of the cardiac vectors as a possible factor. For example, while in young persons these vectors have a relatively longitudinal direction, they acquire a more or less lateral direction in middle age as a result of which the wave amplitude on an ordinary longitudinal ballistocardiogram is reduced. Moss (1961) himself rejects the idea that the changing position of the heart has anything to do with the appearance of age-connected ballistocardiographic changes. He attributes them to the changes in myocardial contractility and vascular elasticity which develop with age. That the changes in the position of the heart cannot be of decisive importance is in some way indicated by the observations made by A. A. Talakov (1958) and A. I. Batrak (1961). A ballistocardiogram of a dextrocardia is not different from a ballistocardiogram taken of persons with a normal arrangement of their organs.

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As for the importance and evaluation of the age-connected changes recorded on a ballistocardiogram, Starr (1947 and 1957) makes a unique observation which is very interesting from a clinical point of view. It is based on a few observations of test subjects above 40 years of age, 8-10 years after their first examination. Starr believes that the presbycardial phenomena are indicative of a symptomless coronary disease characterized by a "subclinical" course. He bases his claim on the following. First, the frequency of pathological ballistocardiograms and the frequency of pathoanatomical information indicative of a coronary disease are practically the same in similar age groups. Second, the use of nitroglycerin by test subjects who have presbycardial symptoms results in a complete or partial restoration of their normal ballistocardiographic picture. Third, a number of the test subjects eventually develop a complete clinical picture of a coronary disease. Fourth, in some cases the autopsy of the diseased patients reveals symptoms of a coronary disease. All these data prompt Starr to conclude that a pathological ballistocardiogram of healthy people over 40 indicating changed wave forms or decreased wave amplitudes has an ominous prognostic significance. A. I. Gefter (1958) holds a similar opinion. Scarborough, Smith and Baker (1960) do not agree with these data, as they

find no biochemical changes in the blood that would characterize coronary disease in the test subjects with age-connected ballistocardiographic changes.

We believe that all the mentioned factors have something to do with the moderate age-connected changes on the ballistocardiogram, but the relatively more pronounced changes should be attributed primarily to latent coronary insufficiency.

Reference to the connection between age and the ballistocardiogram brings up one important practical question. The subject under discussion here is the establishment of normal ballistocardiographic indicators. Is it possible to determine the common norms for all ages, or should they be determined separately by age group? Starr (1958) calls this a "philosophical" question because if common norms were used as a criterion, many healthy people in advanced middle age would then be considered unhealthy. On the other hand, if the norms were applied to individual age groups, the healthy persons who show no age-connected changes on the ballistocardiogram would be considered beyond the range of the particular norm. Starr proposes the use of uniform norms based on the ballistograms of healthy young test subjects. Other authors prefer to have the norms applicable to different age groups so as to have the age criteria for normal and pathological curves. We believe that the establishment of norms for individual age groups would be the proper and logical approach of the clinical physician who has to observe any biological phenomenon within the dynamic pattern of its development. Such an approach disposes of the proposal made by Scarborough et al. (1952) to the effect that persons over 60 should be kept under observation if a normal ballistocardiogram is recorded at that age. On the other hand, it may be possible to have uniform norms but the evaluation of the resulting data should be based on the already known age-connected fluctuations of certain individual characteristics. We have been using this approach in connection with numerous investigation methods. /265

We have not discussed the characteristic features of ballistocardiograms made of children. M. A. Zhukovskiy (1959) succeeded in recording ballistocardiograms of children aged 2 years and up. He finds that a ballistocardiogram of a child is not basically different from the curve of an adult. The only difference is a more pronounced diastolic wave and a higher wave amplitude. T. P. Novikova (1961) notes that healthy children between 4 and 6 frequently show a high, notched H wave that blends with the previous waves, and children between 7 and 14 reveal a high notched L wave and a rounded K wave.

X. THE BALLISTOCARDIOGRAM AND SMOKING

It is a well known fact that smoking may produce certain changes on the ballistocardiograms of healthy people, and in certain cases the changes are quite pronounced. These changes do not last long, usually only a few minutes. The information obtained by various authors shows convincingly that after smoking even a single cigarette, young people and especially old ones,

can change a normal ballistocardiogram to a pathological one. Simon, Iglauer and Braunstein (1954) failed to find such changes or a reduction of the stroke volume (calculated on the basis of the ballistocardiogram) under the effect of smoking up to the age of 40, but Buff (1955) finds an abnormal reaction in 15% of the cases. The changes on the ballistocardiogram amounted to primarily an increase in the H wave amplitude, a reduction of the I wave, a notching and reduction of the J wave, an increase in the respiratory rate, and in a number of cases an increase in the L wave amplitude. The mentioned pathological changes are particularly clearly manifested in patients suffering from hypertension and coronary insufficiency. Fig. 103 shows a noticeable deterioration of a ballistocardiogram recorded of a patient suffering from coronary insufficiency and frequency seizures of angina from smoking.

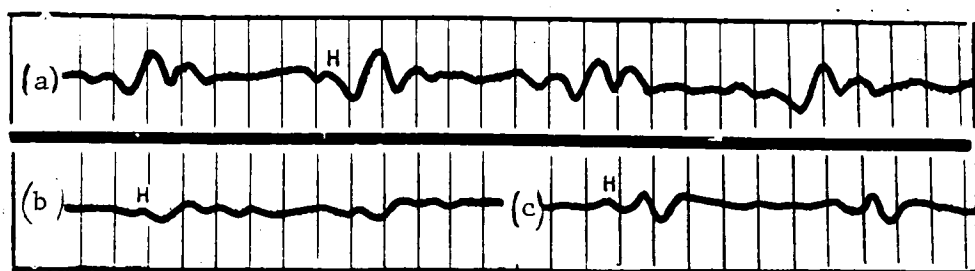


Fig. 103. Ballistocardiogram and smoking. Ballistocardiograms of patient K. S., age 47, with following diagnosis: atherosclerotic heart disease with frequent anginal seizures. (a) Before smoking: second degree curve, based on the Brown method, with pathological wave and interval changes; (b) one minute after smoking one cigarette: a sharp drop in the amplitude of all waves, and the curve approaches the third degree as defined by Brown; (c) ten minutes later: some increase in the wave amplitude is noted.

The effect of smoking on a ballistocardiogram is so characteristic that a cigarette test has been proposed as a functional method of examination, particularly one designed to detect the latent forms of coronary insufficiency. The test is considered positive if the ballistocardiogram reveals changed wave forms, a reduction of their amplitude and intensification of respiration characteristics in the very first minutes after the smoking of a cigarette. Whenever a test of healthy people proves positive, Buff (1955) suggests that they give up smoking without fail. Russek, Zohman and Dorset (1955) discovered the deterioration of the ballistogram under the effect of that test in 50% of the patients with clinical symptoms of coronary insufficiency. Davis et al. (1956) applied that test to both healthy people and those suffering from a coronary disease. Before the test, the ballistocardiogram was pathological in 25.3% of the healthy persons and 56.4% of the sick. The cigarette test was found to be positive in 7.5% of the cases in the first group, and 48.3% in the second group. It is interesting to point out that the use of nicotine-free or filter cigarettes produced no changes on the ballistocardiogram. G. Ye. Tsintsadze (1960) finds that a ballistocardiogram taken of patients suffering from a coronary dis-

ease deteriorates under the effect of the cigarette test, and that the ballistocardiograms showing a more or less normal picture of the patients before the test invariably deteriorate after the cigarette test. I. T. Stukalo, Yu. V. Kulachkovskiy and G. Ye. Shakhinidi (1958) attach great importance to this test in diagnosing the latent forms of coronary insufficiency. /267

The mechanism of the pathological changes occurring on the ballistocardiogram under the effect of smoking has not yet been fully ascertained. Dock, Mandelbaum and Mandelbaum (1953) assume that the stenosis of the coronary arteries and peripheral arterioles as well as the retention of the blood in the veins of the celiac region apparently play an important part in that. The experimental observations made by Corbascio and West (1960), however, show that the local or general use of nicotine or its inhalation produces a positive inotropic effect and improves the ballistocardiogram, while Bellet, West and Guzman (1960) find that the intracoronary introduction of nicotine evokes both parasympathetic and sympathetic responses. A study of certain indicators of myocardial biochemistry in clinical conditions and the catheterization of the coronary sinus by Barger et al. (1957) revealed that smoking intensifies the blood flow in the myocardium of healthy individuals. A detailed investigation prompted Regan, et al. (1961) to conclude that smoking by healthy test subjects or patients with coronary insufficiency increases the heart rate, raises the systemic blood pressure and intensifies the work of the left ventricle, but does not produce any noticeable change in coronary artery blood flow. The authors emphasize that smoking does not develop ischemia in the myocardium, the general belief to the contrary notwithstanding. This is also indicated by Russek, Zohman and Dorset (1955) who believe that the ballistic changes occurring after smoking are not connected with the changes in the coronary system but primarily with the constriction of the peripheral vessels.

XI. BALLISTOCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE SYNTHETIC ELECTROCARDIOLOGIC COMPLEX

Ballistocardiography fills the wide gap which has been left open in the clinical study of individual cardiac functions by electro- or vectorcardiographic investigations. The subject under consideration is the contractile function of the myocardium. The latest available methods can only play an indirect part in determining that function. We do not deny the possibility of determining the rate of cardiac contractions by a ballistocardiographic examination. But this method cannot be very valuable for the study of the various disruptions of automatic cardiac functions. We have already pointed /268 out that, far from being an object of ballistocardiographic study, tachycardia makes such a study impossible. This method cannot play any part nor can it have any indirect or relative importance in the study of cardiac conductivity and irritability, although certain studies appearing in literature do not quite agree with our views. Moss, for example, discovered what he termed characteristic changes in the initial portion of the systolic complex occasioned by a block of the left bundle of His. However, determining the importance of ballistocardiography for the study of cardiac automa-

tism, irritability and conductivity, L. B. Andreyev (1959) reaches a conclusion, similar to ours, that disruptions of these functions are reflected on the ballistocardiogram if they change the hemodynamic conditions directly or indirectly. Consequently, ballistocardiography records not so much the graphic picture of such functional disorder as the disruption of the blood circulation dynamics. For example, the increasing wave amplitude of the post premature ventricular contraction complex is connected with the greater ventricular filling, and the changes occasioned by bundle branch blocks are due to the asynchronism in the intracardiac hemodynamics.

Thus, ballistocardiography is a method used in electrocardiology for the purpose of studying the contractile function of the heart. In this sense, the given method has introduced a functional principle and a number of new physiological concepts on a wide scale into electrocardiology. Starr (1957) points out that such clinically important concepts as cardiac fatigue, myocardial weakness, diminishing cardiac reserve strength, etc., which were in the past considered speculative and beyond the range of science, have acquired definite objective importance thanks to ballistocardiography. It is therefore possible to conclude that ballistocardiography is actually one of the successful methods of the functional investigation of the cardiovascular system, particularly if it is accompanied by certain tests. In this sense, it is possible to grasp R. I. Gismatulin's (1959) bold words to the effect that the development of ballistocardiography should lead to the development of a reliable quantitative evaluation of the functional state of the cardiovascular system.

While attaching much importance to this method, we believe that it would be a crude and grave mistake to expect to indicate the specific anatomical injury to the heart. In this respect it is impossible to approve the course of ballistocardiographic development which is designed to find a characteristic curve for every individually described part of the heart. Such a course may have been consistent with certain rules in the initial development of this method, although Starr has already mentioned in his famous Harvey lectures that ballistocardiography merely tells you whether the heart is strong or weak and how well it performs its blood-expulsion function. We ^{/269} have reached the firm conviction in this work that, with the exception of very rare cases, there is no typical or characteristic ballistocardiograph of a particular cardiac disease or syndrome. Similar pathological changes in the various pathological complexes are found in a great variety of diseases, depending on the functional state of the cardiovascular system. A similar opinion has been expressed by Fidler, Bhargava and Parent (1958), Ye. B. Zakrzhevskiy and R. M. Bayevskiy (1959), etc., as well as by A. H. Kokosov (1959) who believed that ballistocardiography is one of the indicators of the working capacity of a cardiac patient. To prove the validity of our opinion, we will cite briefly the changes of the ballistocardiogram produced by a number of cardiovascular diseases.

T. A. Zubarev (1956) finds that rheumatic heart disease produces a notched and split L wave, O. D. Kuznetsova (1960) sees nothing that is characteristic of rheumatic heart disease, and M. A. Zhukovskiy (1959) finds an increased H wave, diminished and distorted systolic waves and sharp changes

in the diastolic waves in children. Our observations (Z. L. Dolabchyan and M. A. Yesayan, 1961) reveal that reumatic heart disease increases the R-H, R-I, R-J, R-K and R-L time intervals. The increase of these intervals is primarily due to the increasing R-H interval which is indicated by the fact that the intervals between the individual waves of the ballistocardiographic systole frequently remain within the norm, not counting the slight delay of the I wave in some patients. The frequency and extent of the increase in the ballistocardiographic systole, are almost the same as those of the R-H interval. That means that the elongation of the hemodynamic systole is primarily due to the increase in the stress phase, and the intrasystolic indicator is correspondingly reduced. Some increase in the IJ and KL waves is frequently noted (the increase in the IJ wave is more frequently observable in the diastolic period, and the KL increase in the acute state); on the other hand there is also a tendency of the JK wave to decrease. The active phase is characterized by some increase in the JK/IJ coefficient, and the diastolic period by a reduction of the KL/IJ coefficient. The respiratory coefficient is larger during the diastolic period. There is slight or average degree of Brown-defined deviations.

Izak and Braun (1956) as well as Morret et al. (1957) and P. A. Vartapetyan (1960) do not find anything characteristic of mitral stenosis, while V. M. Poznyak (1959) believes that the lower HI/IJ coefficient and the higher JK/IJ and KL/IJ coefficients and the changed H, I and J waves are characteristic of the given disease, and Davis et al. (1953) call attention to the split J wave. Henderson (1955), on the other hand, attaches major importance to the HI wave, and V. L. Karpman and G. V. Sadovskaya (1957) emphasize the significance of the increased R-H interval. It is interesting to note that in the examinations made long after a mitral commissurotomy, K. V. Zvereva (1958) discovered changes corresponding to the changes of blood circulation before the operation. We have discovered slight degrees of Brown-defined deviations in patients suffering from mitral valve insufficiency. Relatively large changes were found in cases of mitral stenosis involving some retardation of the J wave because of the retardation of the right ventricular systole. It is interesting to note that atrial flutter is frequently accompanied by the occurrence of a H and G wave but such changes are not always found. The general structure of the complexes in this case largely depends on the duration of the diastole of the preceding cardiac cycle (Fig. 96). An aortic valve insufficiency reveals an increasing systolic wave amplitude. A similar picture is occasionally observable also in aortic stenosis. Yu. D. Safonov (1959) believes that a split H wave is characteristic of mitral valve insufficiency, a split I wave of mitral stenosis and a changed J wave characterizes aortic valve insufficiency. According to A. I. Batrak (1959), a high IJ and JK wave amplitude, reduced or missing H and I waves, an acceleration of the Q-I interval and elongation of the Q-J interval are characteristic of aortic valve insufficiency. R. M. Bayevskiy (1962) also finds a considerable increase in the amplitude of all the waves, especially IJ and JK, in cases of aortic valve insufficiency. The IJ wave is decreased, K is shortened and M and N are reduced in amplitude in cases of aortic stenosis.

Tabo notes a flattened I wave, a recessed K wave and early M-shaped

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complexes in hypertension, while Lin-Cheng (1957) and Ye. V. Erin (1960) call attention to the increasing R-H interval and diminishing H-K interval, associating the former phenomenon with arteriosclerotic heart disease and the latter with aortic atherosclerosis. I. Ye. Oranskiy (1958) attributes the changes on the ballistocardiogram in the II-III stages of disease to arteriosclerotic heart disease, and from that point of view attaches great diagnostic importance to tests involving exercise and nitroglycerin. Ye. R. Sidorenko and G. I. Sidorenko (1958) note that the progressing disease is accompanied by a systematic increase in pathological deviations which are not characteristic of hypertension. Our observations have revealed an increase in the R-H interval, some elongation of the I-J, H-K and I-K intervals, a reduction in the amplitude of the systolic waves, particularly JK and KL, an occasional increase and splitting of the H and I waves and pronounced postsystolic M and N waves. Various degrees of Brown-defined deviations are observable, but a gradual deterioration of the curve pathology is not always found to accompany the progressing disease. The elongation of the mechanical systole is produced by the increasing tension phase which accounts for the reduction of the intrasystolic indicator; the increase in the R-H interval is more frequent and considerable in the later stages of the disease, and is associated with the weakening contractile capacity of the myocardium and rising diastolic pressure in the aorta. Brown et al. (1952), Rager, (1957), etc., point to the deep K wave in hypertension which is conditioned by high peripheral pressure and the rigidity of arterial walls. We have not always observed that and, furthermore, we have frequently noted a shortened K wave and even an amputated one which accounted for the reduction of the JK/IJ and KL/IJ coefficients; such a picture is associated with the contractile capacity of the myocardium and the hemodynamics as a whole. /271

Many studies of the importance of ballistocardiography for coronary insufficiency are found in literature. In such cases, V. V. Parin and A. V. Mareyev (1954) find mostly some reduction of the wave amplitude, an increase in the respiratory variations, the appearance of blended H-J waves and a higher H wave amplitude; in such patients I. F. Kononenko and N. I. Shtel'makh (1957) find the unification of the H and I waves, a low amplitude, a notched HI, a split I, and increased diastolic complexes. S. S. Belousov (1958 and 1959) discovered, in addition to other changes, a reduced H-K which is connected with coronary atherosclerosis and can be improved after a prolonged treatment. Z. I. Yanushkevichus and G. A. Vitenshteynas (1959) attach some importance to the increasing H wave in case of coronary artery disease, and R. M. Bayevskiy (1962) usually finds early or late M-shaped complexes, a reduced IJ amplitude, intensified respiratory variations and morphological changes during inspiration. L. M. Rakhlin and B. Z. Akkerman (1958) succeeded in differentiating angina-like pains from coronary artery disease in electro- and ballistocardiographic examinations, even though the authors do not find the ballistocardiographic picture characteristic of coronary artery disease. I. L. Manuylov (1958 and 1959) also attaches a great deal of diagnostic and prognostic importance to this method in connection with coronary insufficiency, but cannot find anything specific on the ballistocardiogram of such patients, and R. I. Mangushev (1960) attributes the beneficial effect of tiphen (diphenylthioacetic acid 2-

diethyl aminoethyl ester; a spasmolytic) to the improved contractile function of the myocardium as a result of an intensified coronary blood flow. Studying the quantitative indicators on ballistocardiograms of patients suffering from coronary insufficiency, A. V. Mareyev (1959) points out that they merely provide an objective idea of the contractile myocardial functions of the ventricles.

Many researchers are now studying the changes on the ballistocardiogram produced by myocardial infarction (V. V. Parin and A. P. Matusova 1954; B. Z. Akkerman, 1957; A. P. Matusov, 1959; A. L. Limcher, 1960; Starr and Wood, 1943; and Scarborough et al. 1952; etc.). The papers submitted by the mentioned authors describe a variety of changes (a considerable reduction of the systolic wave amplitude, various deformations of individual waves, changing respiratory variations, pronounced Brown-defined deviations, etc.), especially in the initial period following the development of an acute seizure which eventually disappears with the improvement of the patient's condition. In this sense, ballistocardiography is prognostically important in cases of myocardial infarction, especially since electrocardiographic changes remain for a relatively long period of time while ballistocardiographic changes, given a favorable course of the disease, go back to normal in a relatively shorter time. Of some interest is the opinion held by Dock, Mandelbaum and Mandelbaum (1951); they believe that if the myocardium is in good condition, the ballistocardiogram may be normal even after the infarct. Such a view is understandable as the ballistic forces are generated more by the movement of the blood rather than the fluctuation of the ventricular muscle (Dock and Grandell, 1957).

The importance of ballistocardiography in an early diagnosis of coronary insufficiency is emphasized in literature. We have studied that problem. Under observation was a group of patients with more or less pronounced painful syndromes in the cardiac region resembling coronary artery disease but a detailed objective examination in these cases, including electrocardiography, failed to disclose any definite changes that would indicate a pathology of the coronary blood circulation. The majority of the patients revealed pathological curves of IIb degree or higher, and the wave deformation was primarily occasioned by the reduction of their amplitude, the splitting of the J and K waves and the appearance of early M-shaped complexes. In these observations the age factor is almost irrelevant, as the average age of the patients even with a IV degree pathology was not more than 50. We believe that the deterioration of the ballistocardiogram is indicative of a latent disturbance of coronary blood circulation. We did not find any specific changes characteristic of coronary insufficiency. However, there was still a direct connection between the detected ballistocardiographic changes and coronary insufficiency. Disrupting the circulation in the heart, the latter is conducive to changes in the myocardium, a reduction of the cardiac functional capacity and disruption of the hemodynamic systole. Hence, the conclusion that latent forms or early manifestations of coronary insufficiency can be detected through the hemodynamic systole earlier than through the electrical systole.

We consider it unnecessary to discuss the studies dealing with the im- /273

portance of ballistocardiography in other diseases, such as chronic pulmonary diseases (Yu. A. Panfilov, 1957 and 1961; Yu. I. Detsik 1961), scleroderma (F. S. Drampyan, K. A. Kyandaryan and Z. L. Dolabchyan, 1962), acute anemia (Sanghvi and Banerjee, 1959; Tandon and Katiyar, 1961; A. D. Yanovskiy, 1959), diabetes mellitus (Rakel', Skillman and Braunstein, 1958), endemic goiter and thyrotoxicosis (M. E. Vasil'yevskiy and Ye. N. Dormidontov, 1958), Botkins disease and acute dysentery (V. A. Anisimova 1959), brucellosis (V. I. Leyman, 1959), etc. The changes in all these diseases are connected with disrupted myocardial functions because of the effect of the diseases on the heart.

This confirms the principle that a ballistocardiographic examination cannot play any substantial part in an anatomical diagnosis of a particular cardiac disease or syndrome, but it provides an idea of the contractile capacity of the heart in these disturbances. Ballistocardiography, in other words, is a nonspecific method of examining such diseases. This should not be construed to mean that we are allegedly attaching specific importance to electro- or vectorcardiographic methods. This is not what we have in mind, but the latter methods, by revealing a particular syndrome or disrupted function, contribute to the establishment of an anatomic diagnosis. At the same time we do not deny the importance of such methods in the function of diagnosis of cardiac diseases.

In the light of this information, it becomes necessary to define the relationship between electro- and ballistocardiography. To begin with, the first method is used to study the electrical activity of the heart, and the second the mechanical activity. Furthermore, it should be emphasized that the object of electrocardiographic examination is the heart as an integral organ, which is almost isolated from the other components of the circulatory system. Ballistocardiography deals with the function of the entire cardiovascular system, as the formation of the ballistocardiographic curve is to a large extent determined by the function of the vascular system. Secondly, the individually described diseases are in various degrees reflected on the electrocardiogram, and an experienced electrocardiologist is frequently able to make an assumption of the type of disease on the basis of the electrocardiographic data alone. This is not possible in the interpretation of the ballistocardiographic data. Thirdly, ballistocardiography is a more sensitive and more labile method than electrocardiography, and it very rapidly reacts to changes in the functional condition of the heart in a particular direction. This can be understood from the following. Our observations show that the electro- and ballistocardiographic data on the same test subject are not always similar, and that occasionally there is even some contradiction involved. This seeming contradiction will be explained if we point out that ballistocardiography as a method of functional diagnosis may produce information which does not quite reflect the anatomic condition of the heart, while electrocardiographic data reflects the anatomic picture of the heart to a more or less high degree. It should be assumed that the issue under consideration in this case is the relationship between the organ and its function, that is, the mutual relation between relatively larger biostatic and biodynamic phenomena. The clinical physician realizes that similarity between these two aspects of a single whole is not always an invari-

able pattern.

In view of these considerations, it becomes clear why in some cases the repeated ballistocardiograms taken of the same test subject reveal somewhat different pictures. Such a variability of the ballistocardiogram is due to the great lability of the mechanism of hemodynamics and not to any technical defects of the method, as some authors believe. In this respect we agree with Talbot (1958) that a more profound study of the mechanical factors of cardiac contraction and blood circulation as a whole is required in order to prevent technical errors that might affect the prestige and importance of this valuable method.

XII. CONCLUSION

All the above considerations justify the conclusion that ballistocardiography is a good electrocardiological method of studying the most important aspect of cardiac activity, that is myocardial contractility, and learning about the functional condition of the cardiovascular system. In this sense it is an important supplement to electrocardiography, and it has its special place in synthetic electrocardiology. It is our opinion that the further development of this method should not be designed to search for "specific" changes on the curve representing a particular disease, but to detect the ballistocardiographic symptoms which reflect a particular functional condition of the heart and a particular degree of the disrupted propulsive capacity of the myocardium. It is from these positions that we must understand and judge the significance of the efforts being made in the study of various cardiac drugs (A. M. Kochetov, 1958; Malt, 1958), or the recommendations to use ballistocardiography for the mass examination of certain groups of population (R. M. Bayevskiy, 1959), or for the prophylactic examination of the middle-aged and elderly population (H. K. Furkalo, 1960), for determining the best period for a mitral operation (K. V. Zvereva, 1960) or establishing the type of congenital disease (T. P. Zaitseva-Gryaznova and S. S. Belousov, 1960), for the use in sports medicine (L. I. Stogova, 1956 and 1959; B. Kushelevskiy and A. Kokosov, 1958; V. V. Barabanshchikov et al. 1959), for the study of therapeutic factors (A. A. Romanova, 1957; E. A. Tambiyev, 1959; I. Ye. Oranskiy, 1959; V. D. Dzhordzhikiya 1960), or for the detection of various factors in occupational pathology (A. I. Levin, 1959). Such an approach makes it clear why entirely different pictures are frequently recorded on different patients suffering from the same disease, while, on the other hand, the curves recorded of different diseases are quite similar. The decisive factor in this case is not the type of the anatomic injury to the heart, but the contractile capacity of the myocardium and its hemodynamic systole.

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We do not attach any importance to ballistocardiography in connection with the quantitative determination of the stroke volume, although we find that it can provide some idea about it, especially in dynamic investigations.

We wish to finish this chapter with the following words expressed by Starr (1958): "...Ballistocardiography reveals an aspect of cardiac activi-

ty which is just as important as blood pressure, and if physicians learn of its possibilities and acquire the habit of using the information it contains, its value will be just as great as the knowledge of blood pressure is today."

CHAPTER II

PHONOCARDIOGRAPHY

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I. INTRODUCTION

In spite of the fact that Hippocrates had already spoken in his "de Morbis" about direct auscultation of the chest and that Harvey in his classical works described the heart sounds, auscultation of the heart was introduced in medicine as a most important method of heart examination only when Laennes, in 1819, submitted his stethoscope and used it for description of the tones and noises produced by the heart. During its one hundred and forty years of life, auscultation of the heart has proven itself to be of great value in all branches of medicine, particularly in cardiology, and the stethoscope became a constant companion of every physician.

With all of its advantages, however, heart auscultation has certain definite defects. In the first place, an important role is played by individual auditory peculiarities in acoustical perception. This leads to differences of opinion even among specialists regarding determination of the character of discernible acoustical phenomena (such as their intensity, frequency, and location within the cardiac cycle). In the second place, from a physical standpoint, the human ear cannot qualify as a perfect apparatus. Thus, for example, the heart tones may assume frequencies below 16-20 cps. The human hearing apparatus is very sensitive to high frequency sounds emitting from the heart; perception of low frequency sounds is much less sensitive. Thus, while maximum sensitivity of the human ear is for a sound frequency of 2000 cps, it is 50% lower for a frequency of 1000 cps and only 1% of the maximum for a frequency of 100 cps. The human ear can more easily perceive variations in tonal pitch than in tonal intensity. But, again, sensitivity with respect to pitch is lower in the region of low frequency vibrations. The low frequency sounds are of greater intensity and therefore hinder (masking phenomenon) perception of higher frequency sounds which are more important so far as information is concerned. Sounds of equal intensity, but of different frequencies are perceived as sounds of different volumes. /277

These and other defects of the human hearing apparatus, as well as a certain subjectiveness in the use of the auscultating method, necessitate the need for an objective method of recording the sound phenomena of the heart. The first attempts in this direction were made in 1893 by Hurthle who used innervated frog muscle as a sound registering apparatus. In 1894 Einthoven and Geluk used a capillary electrometer and recorded their data as a function of time on the curve obtained. In 1904, Frank suggested a pneumatic method, where the sound waves were transferred from the stethoscope to a capsule and caused oscillations of a mirror which in turn, were registered by means of a projection system. Somewhat later, Einthoven in 1907, developed a method of recording sound vibrations by means of a string galvanometer. Later, many other authors worked intensively and resource-

fully in this area.

The phonography of today is based on solid theoretical and practical considerations and renders a great service with respect to exact quantitative analysis of various cardiac melodies and for explanations of a whole series of hemodynamic phenomena. This method refines and supplements the data obtained by the auscultation of the heart. This is discussed in a monograph by Calo (1938, 1950), Weber (1956), Holldack and Wolf (1958), Lian (1961), L. M. Fitileva (1962), and other authors. The main advantage of phonography as compared to the auscultation method consists of the fact that phonography makes possible an exact determination of intermittent correlation of tones or noises of the heart. Evans (1951) feels that phonocardiography should not serve for detection of those sonic phenomena which are impossible to discern during ordinary auscultation. We find, however, that phonocardiographic study should certainly be conducted in parallel with the auscultation of the heart in order not only to refine the character of the sonic phenomena under investigation, but also to make apparent the tones or noises which are not perceived by auscultation.

II. TECHNIQUES OF PHONOCARDIOGRAPHY

1. Physical Principles

By definition a sound is an adequate stimulus of the auditory analyzer, which is generated as a result of wave-like oscillations of an elastic medium. /278 Any substance - solid, liquid or gaseous - subjected to mechanical vibrations of frequency and intensity lying within certain boundary limits, becomes a source of sound. The audible sounds encompass the region from 16 - 20 to 20,000 cps; sounds below this frequency (the so-called infrasounds) or above it (the ultrasounds) are inaudible.

The shape of a sound wave is a combination of sinusoidal oscillations, the lowest of which is considered to be basic, while the others are regarded as the harmonic overtones. The propagation of oscillations is spherical, and a propagation through a heterogeneous medium changes considerably the initial physical properties of sound. The laws of propagation of a sonic wave from its source to the perceiving organ depend on the peculiarities of the source itself as well as on the characteristics of the transmitting medium. Every sound has three basic characteristics: pitch, intensity and timbre. The pitch depends on the number of oscillations per second. The more frequent the sonic oscillations the higher is the pitch and vice versa. The intensity of sound is directly related to the amplitude of vibrations. In practice, it is preferable to measure the relative intensity. This measurement is expressed logarithmically in whole numbers (bels) or their tenths (decibels). The timbre is the quality of the sound or its tonal hue which depends on the character, quantity and sequence of generation of the overtones. The volume is a complex function of the intensity of sound and of its frequency. Low pitch sounds of equal intensities are of a lower volume than high-pitch sounds. Actually, the volume is a degree of hearing perception. The duration of a sound depends on the frequency of appearance and disappearance of oscillatory motions.

In contrast to tones, noises consist of random, irregular oscillations devoid of regular wave-forms and periods. Such a mottled physical pattern is intensified by the appearance of a discord of overtones.

In a functioning heart, the vibratory oscillations are generated as a result of the rythmical action of the valve apparatus, by contractions of cardiac muscles and by hydraulic factors of the internal cardiac dynamics. These oscillations propagate to the surface of the chest cage through the media of the surrounding tissues. Among these manifold and diverse oscillations there are those which are below the range of the human ear. Such oscillations are of no acoustical significance and therefore are not considered either in the auscultation or phonocardiography processes. The more frequent chest oscillations are perceived as tones and noises. They comprise the "melody" of the heart. All sonic phenomena related to heart activity, from a physical standpoint, are nothing but noises. They differ from one another only by their duration. Thus, the normal sounds of the heart (the tones) are of short duration. The pathologic noises are of considerably longer duration. From this standpoint, it is logical to accept /279 the proposition of French authors distinguishing the physiological noises (the normal tones) from the pathological ones.

The tones of the heart have a frequency from 50 to 100 oscillations per second, while the noises may have 600 or even 1000 oscillations in one second. Rappaport and Sprague (1941) assert that no acoustical phenomenon produced by cardiac mechanism can have more than 1000 oscillations per second. Caniggia, Bertelli and Lodone (1961) determined, by increasing the speed of the paper, that the frequencies of the majority of noises occurring with mitral or aortic stenosis, with that of the pulmonary artery and with certain defects in the interventricular partition, are less than 150 oscillations per second. The noise caused by the inadequacy of the aortic valve has a frequency of less than 250 oscillations per second, while the musical noises have frequencies from 300 to 350 per second. A spectrophonocardiographic analysis shows, however, that the frequency spectrum of systolic and diastolic noises reaches the value of 800-1000 cps with well defined frequencies up to 640 cps. (L. N. Goncharova, 1962.)

2. Techniques

The technique of any phonocardiographic apparatus consists of an ability to perceive and to register all sonic oscillations occurring during the activity of the heart. The apparatus must reproduce only the sonic phenomena, while eliminating all possible fluctuations of the chest frame partition (Calo, 1958), or, more correctly, as it is recommended by the bureau of phonocardiography standardization, the apparatus must register the mechanical vibrations produced by the heart and the neighboring vessels. (Mannheimer, 1957). This means that, schematically, a phonocardiograph must consist of a perceiving apparatus for discerning the mechanical oscillations, of a transformer to transform these mechanical phenomena into corresponding electrical equivalents, and of an electronic unit designed to amplify the electrical phenomena for their registration. This scheme includes also the devices for transmission and amplification of only those sounds which are

within the required range of vibratory frequencies.

Phonic oscillations are discerned at the chest frame partition by the use of microphones which simultaneously perform the role of the transformers. The microphone is an electroacoustic device transforming mechanical oscillations into electrical phenomena. These are amplified and transmitted to the registering apparatus. Of the various types of available microphones, only those of a crystal type are used in phonocardiology. The principle of such microphones is based on the piezoelectric effect. Inside of the microphone there is placed a crystal (quartz, Seignette's salt, barium titanate), which, when subjected to mechanical sound vibrations, generates an electromotive force whose characteristics depend on the frequency and the amplitude of these vibrations. /280

Two types of crystal microphones are used in the phonocardiographic process: the stethoscopic type and the logarithmic type. We shall not consider here the third type - the so-called "linear microphone," because it amplifies equally all oscillations of the chest frame. As a result, the oscillations of heart sounds are almost lost in the background of large amplitude waves corresponding to chest oscillations of the pre-sonic frequencies. This type of microphone is used for registration of cardiograms, arterial sphygmographs or precordial ballistocardiograms.

A stethoscopic microphone intercepts all oscillations of the chest partition except those of very low frequencies (0-10 cps) which are of no acoustical significance. In this manner there is obtained an oscillogram of sonic vibrations in the form transmitted by the stethoscope to the auditory apparatus of the investigator. The resulting curve gives a general idea of the phenomena taking place within the ear of the physician during the auscultation of the heart. Simultaneously, the study is made of both relatively high and relatively low frequency vibrations and the result obtained corresponds very closely to the normal conditions of the Cochlear auscultation (Brandes, 1958). Such a microphone is used for a complete study of phonic phenomena of the heart.

A logarithmic microphone, like a human ear, logarithmically distorts sonic vibrations and, as noted by Donovan, produces data which are comparable to the sonic perceptions of an experienced specialist during the process of auscultation. With the use of such a microphone, the effect of low frequency vibrations is very much decreased, therefore, it can be used for the registration of high frequency noise vibrations.

Contemporary phonocardiographs are constructed in such a manner that a single microphone can register various frequency components of heart sounds. The use of a multichannel apparatus permits the possibility of synchronous registration of these curves on different channels. A self-writing apparatus has certain technical advantages, but it cannot be used for detailed scientific analysis, because under conditions of frequent oscillation of the cardiogram, the inertia factor of the writing device may produce a distorting effect. Brody, Frazier and Lowe (1960) find, however, that after certain technical modifications the self-writing apparatus can be used very successfully.

Contemporary apparatus are supplied with electric filters for a selective reducing or increasing of their sensitivity with regard to preassigned frequencies or vibrations. For this reason Maass and Weber (1952) proposed a "filtered phonocardiography" with the use of five filters: "tief" (35 cps, /281 "mittel 1" (70 cps), "mittel 2" (140 cps), "hoch 1" (250 cps) and "hoch 2" (400 cps).* An analogous principle was used in the construction scheme of Soviet phonocardiographs FKP-1 and FEKP. The Hungarian-made phonocardiograph of type FKG-01 which we now use (as an attachment to the five-channel electrocardiograph of type EKG-01) has three amplifiers with five frequency characteristics encompassing a wide spectrum of heart sounds within the range of 20 to 800 cps. Luisada and his co-workers (1956, 1957) offer a method of "selective phonocardiography," in which the stethoscopic method is used together with two filters - one for vibrations from 60 to 110 cps and the other for those from 150 to 200 cps. The authors recommend this method for investigation of noise phenomena.

Of great significance is the registration of high frequency phonocardiograms. Luisada and Bartolo (1961) find that high-frequency phonocardiography (over 300 vibrations p/s) reproduces very well such phenomena as the splitting of the first and second heart sounds, clicking produced by opening of the mitral valve, and the noises of inadequacy of the mitral or aortic valves.

Calibration causes great difficulties in phonocardiography because many extracardial factors produce an influence on the intensity of the sonic phenomena of the heart. There exist various methods of calibration. Mannheim (1941) introduced a method of "calibrated phonocardiography" which uses filters giving six frequency characteristics of sonic phenomena of the heart. Each of these characteristics is amplified separately according to the principle that low frequency heart sounds (which are essentially normal) require a comparatively small amplification in comparison with their overtones and the phenomena of noise. Luisada and Gamma (1954) offer an original method of calibration, by means of producing a sound signal on the chest whose amplitude and frequency are known, and of its registration by an adjacent microphone. For this purpose Luisada (1953) places a camera with an electric bell within the area of the left chest musculature: a logarithmic microphone. The bell produces a sound of an intensity of 90 decibels. This sound passes through the corresponding tissues and is intercepted by the microphone. The registered value then serves as a standard of comparison. In the phonocardiograph of the type FKG-01, the principle of calibration with a compensation for sound-absorbing characteristics of the human body is also used. In this case, a portable calibrator is attached to the axillary region, and the microphone is placed at the center of the precordium. We have used a different method for calibrating this apparatus. Following the principle of continuous calibration of the whole apparatus, the microphone

Translator's note: * "tief" in German means low
"hoch" in German means high
"mittel" in German means middle

is arranged over the membrane of the calibrator after the registration of the phonocardiogram, and then the obtained signal is recorded. We find that a determination of the sensitivity of the apparatus requires considerable experience and must be done by visual control, because insufficient or excessive amplification hinders the analysis of the curve obtained. /282

Thus, there exist various principles and methods for the recording of a phonocardiogram. It must be noted that such a situation cannot serve as a good basis for further development of phonocardiography. The necessity for standardization is so great that special conferences in Paris (1950, 1953) and in Stockholm (1956) were dedicated to this very question. This important subject is also studied in the Soviet Union (A. M. Rybakov, 1958). In Holland, a special committee has been organized for standardization. This committee recommends construction of standard equipment (Bekkering and Weber, 1957). Unfortunately, general standardization does not yet exist, and the presence of a great variety of principles and apparatus characteristics only hinders somewhat the broad development of the theoretical and practical questions of phonocardiography.

3. Methods

It is known that during the propagation of sound through a heterogeneous medium (as in the chest), there is a considerable loss of sonic energy. This loss depends on the physical characteristics of the medium (such as its specific weight, elasticity, refraction, etc.). Therefore, it is important to seek such points on the chest surface where it is possible to obtain optimal perception of heart sounds.

Various positions and schemes of microphone arrangement have been suggested for the recording of heart sounds. In our practice we record phonocardiograms at the points of auscultation of the corresponding valves. Thus, the mitral valve is auscultated in the region of the apex or at the fifth point; the aortic valve is auscultated in the region of intercostal II, to the right of the sternum; the tricuspid valve on the level of the base of the xiphoid process on the sternum, or somewhat to the right; the pulmonary artery in the region of intercostal II to the left of the sternum. In addition to this we also make recordings of those areas which are of interest in some particular case. In certain cases we make a recording of sounds from the heart apex with the patient lying immobile on his left side; a microphone is also placed in the region of the zero point (which according to the recommendation of the Institute of Cardiovascular surgery AMN, SSSR, indicates the axillary region on the level of the heart apex). The plotting must be made under conditions of complete immobility of the patient with the respiration arrested during the phase of exhalation. /283 This is of great importance since the respiratory act negatively influences the quality of the recorded diagram and, in addition, may prove to be a cause of distortion of the heart sounds as it changes the anatomical arrangement of the organs and tissues within the chest thus altering the interthoracic hemodynamics. (See page 246, original text). Six or seven complexes must be recorded at each region and the rotary velocity of the membrane must be increased to 75 or 100 mm per second. For a more detailed

analysis of the diagrams obtained, Caniggia, Bertelli and Ladone (1961) suggest cardiography with an increased rotary speed of up to 15 meters per second. We agree with Green and Freeman (1949) that the stethoscopic diagram must be recorded with the aid of a series of electrofilters. The placement of the microphone (for adults we fix it in position with a rubber belt; for children we use hand pressure) must be done under certain pressure in order to obtain a hermetically closed space between the microphone and the chest surface. In the examination room complete silence must be maintained. It is preferable that the room be at a distance from all noises inside or outside the building.

During the process of examination it is possible to follow the sonic phenomena with the use of an audiphone, which transforms electrical waves into sonic and transmits them to the listening apparatus of the investigator. In our practice we use a loudspeaker which permits auscultation by several persons simultaneously. Of great help in this connection may be the method of audio-video-cardiography. With this method, the registration of the cardiac sounds is accompanied by visual observations, auscultation and, when necessary, by recordings on the magnetophonic membrane. (Briskier, 1953). A few words may be said at this point regarding the telestethoscopic method which permits auscultation simultaneously by several persons and at the same time a registration of the audible acoustical phenomena. (Lian, Minot et Welte, 1941). It must be taken into consideration that a phonocardiogram may frequently show certain distortions which are due to such factors as asthmatic respiratory motions, crepitations or distinct respiratory noises, friction between the microphone and the chest hair, muscular tremor, noises from the alimentary tract, nonconformance with the requirements of absolute silence within the examination room, etc. Such distortions are easily recognizable as they appear without any regularity. If they pertain to the respiratory apparatus they appear in cycles corresponding to the respiratory rhythm.

In order to determine the significance and the origin of individual oscillations on the diagram, the phonocardiogram must be recorded simultaneously with indicator whose characteristics are well known and do not give rise to doubts.

As long ago as 1894, Einthoven and Geluk synchronously recorded the phonocardiogram with heartbeat at the apex, or with a sphygmogram of the carotid, and Far in 1913 used the method of superposition of the phonocardiogram on the curve of the electrocardiogram. In our work, one channel of the apparatus is used for graphic II of the electrocardiograph. A greater than usual amplification is used in order to express more clearly the individual components of the electrocardiogram. We often register simultaneously the pulsation of the carotid. Also a good indication is registration of venous pulsation. /284

In addition to the generally accepted method, there also exist phonocardiographic methods for specific purposes. Certain specialists, in the solution of problems connected with the development of the foetus and with the process of child bearing, ascribe a definite significance to the registration

of the heart sounds of the foetus. (Fig. 103A) In this case, a microphone is placed at those points on the abdomen of the pregnant woman where audibility of the heart beat of the foetus is optimal (Sadern, 1954). There are other methods of phonocardiography such as alimentary (Taquini, 1936) and intratracheal phonocardiography. The latter method is of importance for the study of sonic phenomena occurring at the region of the heart base. There exists a synchronous bipolar method of phonocardiography, where the recording is done simultaneously from two different areas. This method results in very valuable information for the study of the second sound in the area of the main vessels. (Leatham, 1952; G. I. Tsintsadze and N. V. Lomsadze, 1961). Especial significance must be ascribed to the epicardial (Bertrand, Milne and Hornick, 1956) and intracardial (Lewis (1957), Wallace (1957), Feruglio (1959), etc.) phonocardiographs. These types will find an increasingly greater use with the rapid development of heart surgery.

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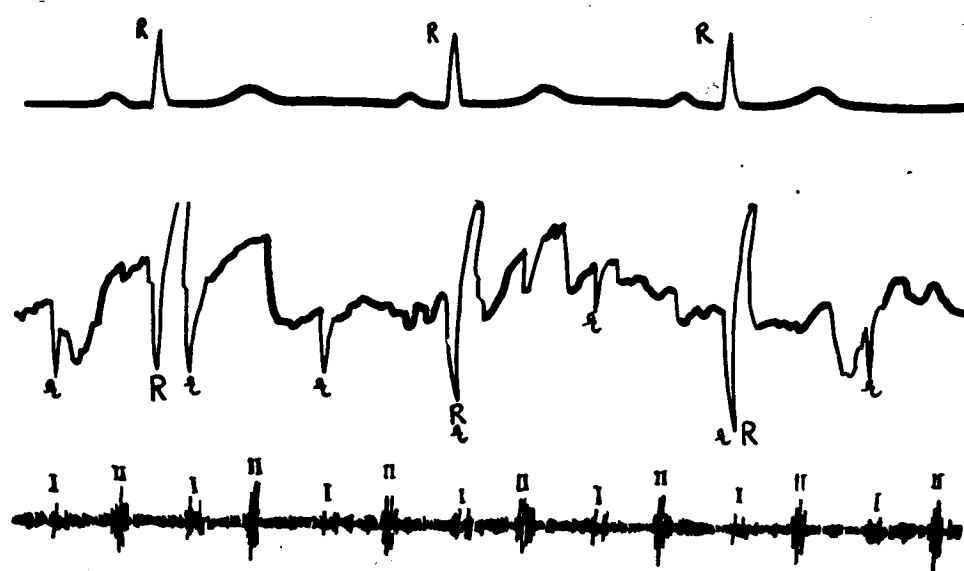


Fig. 103A. Phonocardiogram of the fetus. The first channel registers graphic II corresponding to the electrocardiogram of the mother; the second channel registers the electrocardiogram of the fetus (three electrodes are arranged in a triangular form within the hypogastric area); the third channel registers the phonocardiogram of the fetus (the microphone is placed in the area of optimal auscultation of the foetal heart sounds). R corresponds to the complex of the mother's heart; r to that of the fetus. I - the sound is bifurcated; II - the sound is amplified.

A separate place must be allotted to the original method of Roentgenologic endoauscultation developed so well by I. I. Savchenkov (1954, 1955). The author claims this method of roentgeno-phono-diagnostics to be based on study of the sonic phenomena of the esophagus, trachea, stomach, and duodenum. It presents a method of determining the localization of the foci of sound formation within the heart and vessels.

III. NORMAL PHONOCARDIOGRAMS

Four types of sounds, labelled: I, II, III and IV are distinguished on a normal phonocardiograph (Fig. 104). During auscultation of the heart, usually only types I and II are perceived, which, according to their position, may be identified as the systolic and diastolic tones. Sound types I and II originate during systole and thus are known as normal systolic sounds in contrast to types III and IV which originate during the diastolic phase and are known as normal diastolic sounds.

According to Greene (1957), our conceptions and knowledge regarding the formation mechanics of these sounds was based, until recently, on indirect observations of humans and direct examinations of animals. Due to the introduction of a series of new methods of investigation it has now become possible to study the source of various sounds directly in the interior of the human heart.

The Luisada (1949, 1953) diagram of sound structure is given below.

The first sound is of muscular-valve-vascular origin and forms an intricate complex consisting of five parts. The first part appears synchronously with wave R of the electrocardiogram or a little after it. It is an oscillation of low frequency and medium amplitude. Appearance of this part is connected with initial tension during ventricular systole, but in its formation retrograde fluctuations of the auricular contractions play a very definite role. Grishman and Bleifer (1960) think, however, that the role played by the auricular contraction in the formation of these oscillations is not clear because such oscillations are also registered for patients with auricular fibrillation. The second part consists of one or two strong oscillations caused by the closure of the atrioventricular valves. Braun- /286wald and Morrow (1958) think that sound I starts exactly at this moment. The third part consists of relatively small oscillations caused by vibrations of the cardiac septa or vibrations of the chest. The fourth part consists of one or two oscillations which are developed due to the opening of the semilunar valves of the aorta and the pulmonary artery. The fifth part consists of one or two small oscillations originating in the stretching of the walls of the main vessels during the phase of expulsion.

Comparing the internal pressures within the cavities with the data obtained for intracardiac pressures and for ordinary phonocardiograms, Luisada (1958) comes to the conclusion that the sound I complex can be divided into only three parts (Fig. 104). The first, or initial part, originates as a result of the tension within the myocardium. Its duration is 0.02-0.04 sec. and it consists of low amplitude oscillations. The second, or middle part, is of valvular origin (closure of the mitral and tricuspid valves and opening of the pulmonary artery and aortic valves). It lasts 0.05 sec. and consists of high frequency oscillations. The third, or final part, is of a vascular origin, lasts 0.02-0.04 sec., and consists of low /287frequency oscillations. During auscultation, essentially only the second part is perceived.

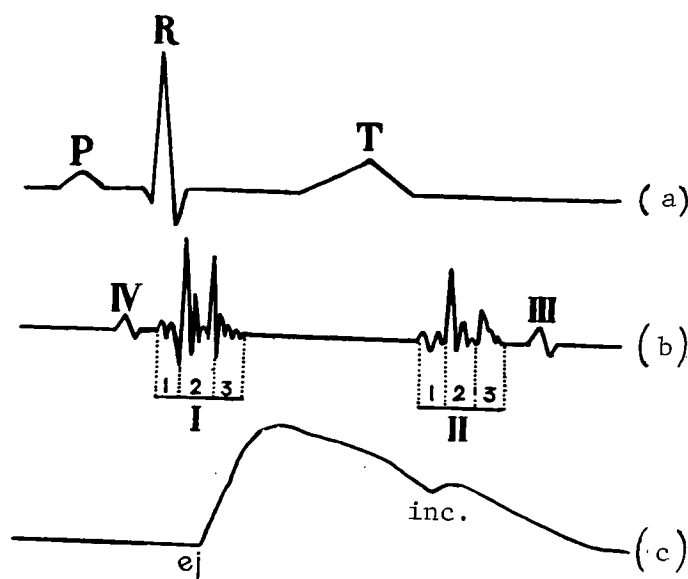


Fig. 104. Normal Phonocardiogram. (Schematic) (a) Electrocardiogram. (b) Phonocardiogram: Sound I and its three parts (1,2,3), Sound II and its three parts (1,2,3); Sounds III and IV. (c) Sphygmogram of the carotid; ej (ejection) - the beginning of the rise of the pulse wave; inc - incision.

In the case of intracardiac phonocardiography, Moscovitz et al. (1960) found two distinct components of the I sound. The first component coincides with the instant of closure of the atrioventricular valves, while the second corresponds to the instant of blood expulsion into the main vessels. The interval between these two components reflects the isometric contraction phase.

The amplitude of the I sound depends on a series of factors governing the energy of the ventricular systole. The importance of the two following factors must be stressed: the duration of the preceding cycle (Meda and Scansetti, 1957), and the position of the atrioventricular valves at the end of diastole (Leatham, 1958). In this respect Levine (1948) notes the fact that the intensity of the I sound is inversely proportional to the length of interval P-R on the electrocardiogram. Also of great interest is the frequency of the I sound.

While comparing ordinary phonocardiograms with those obtained for the alimentary channel, Miller and Groedel (1950) discovered that the I sound pertaining to the alimentary phonocardiogram consists of four broad oscillations with a frequency of from 25 to 50 cps, and is of longer duration than the I sound of the ordinary phonocardiogram. Using phono-spectrometry, G. I. Kassirskiy (1960) discovered that the frequency of the I sound at its apex is 500 cps, while frequencies of the maximum intensity are 178 cps.

Definite significance must be ascribed to the measurement of the interval Q-I, i.e. the interval of time between the depolarization of the ven-

tricles and the appearance of the I-sound. Different norms for this interval are assumed by different authors. In our work we assumed the interval from 0.02 to 0.06 sec. as the normal duration of the Q-I interval.

Four parts can be distinguished in the complex of the II sound. The first part consists of one or two low frequency, small amplitude oscillations, appearing during the diastolic diminution of pressure within the ventricle at the beginning of its isometric relaxation. The second part represents a series of oscillations, usually of high pitch and amplitude. These oscillations are caused by the closure of the semilunar valves; the initial large oscillations correspond to the closure of the aortic valves. The oscillations which follow are relatively slight. They reflect the closure of the valves of the pulmonary artery. The third part consists of one or two low pitch and low amplitude oscillations. They are caused, evidently, by the vibrations of the heart and vessels; Ongley et al. (1960) thinks that they are also due to the oscillations of the chest. The fourth part consists of slight oscillations due to the opening of the atrioventricular valves.

Luisada et al. (1958) purport to distinguish only three components of the II sound (Fig. 104): the initial low-frequency part preceding valve closure; the central high frequency part reflecting the closure of the aortic valves and those of the pulmonary artery, and the final low-frequency part, pertaining to the opening of the tricuspid and mitral valves. During auscultation, essentially only the second part of the II sound is perceived. The intensity of this sound depends on the amplitude of its aortic component. The pulmonary component is very weak, (the ratio of intensities of the aortic and the pulmonary components is 2 to 1) and is usually only localized in the area of the pulmonary artery whereas the aortic component radiates into all areas, particularly into the area of the apex of the heart. /288

The III and IV sounds are not always detectable. They are sometimes detected in children and in flat chested adults between the ages of 30 and 40. It is interesting to note that R. D. Dibner has often found these sounds in solidly built sportsmen. These almost inaudible sounds are of low frequency and have only from 10 to 50 oscillations per second. They are somewhat more audible in the area of the heart apex, especially when the stethoscopic system of registration is used. The III sound consists of one or two slight oscillations ($1/3$ or $1/4$ part of the II sound) and appears at the beginning of the rapid, passive filling of ventricular diastole. It is probably due to the motion of the atrioventricular valves and to a sudden stretching of the ventricular septa. It usually appears from 0.10 to 0.15 sec. after the beginning of the II sound. The IV sound consists of oscillations similar to those of the III sound. Its amplitude is equal to $1/3$ or $1/4$ of that of the I sound. It appears during the phase of rapid, active filling of ventricular diastole and is, evidently, due to the contraction of the auricles. Usually it appears 0.14 sec. after the start of wave R of the electrocardiogram (Frost, 1949). This sound is often fused together with the first part of the I sound and constitutes the auricular component of the I sound.

Table 8

Average Duration of Sounds and Intervals According to
Luisada, Mendosa and Alimurung (1949)*

Age Groups	Sound Duration in Seconds										III	IV	II-III
	The I Sound					The II Sound							
	Full	1st	2nd	3rd	Full	1st	2nd	3rd					
Sound of Foetus Age: Under 4 years	0.085	0.010	0.025	0.055	0.055	0.010	0.027	0.020		-	-	-	
	0.070	-	0.040	0.030	0.060	-	0.020	0.040		-	-	-	
4 - 10 years	0.120	-	0.040	0.080	0.065	-	0.015	0.050		0.050	0.060	0.12	
	1.145	0.020	0.070	0.065	0.110	0.010	0.055	0.050		0.040	0.060	0.14	
11 - 20 years	0.147	0.016	0.069	0.071	0.097	0.018	0.015	0.056		0.050	0.060	0.14	
	0.147	0.010	0.064	0.066	0.120	0.020	0.034	0.056		-	0.055	-	
21 - 40 years	0.146	0.020	0.063	0.078	0.107	0.020	0.028	0.069		0.061	0.064	0.16	
	0.145	0.020	0.060	0.071	0.114	0.018	0.043	0.055		0.043	0.052	0.18	
41 - 60 years	0.149	0.020	0.057	0.080	0.097	0.016	0.024	0.068		0.057	0.061	0.18	
	0.144	0.020	0.064	0.068	0.098	0.013	0.040	0.053		0.040	0.052	0.19	
Over 60 years	0.141	0.024	0.050	0.080	0.087	0.020	0.025	0.053		-	0.050	-	
	0.123	0.023	0.063	0.054	0.085	0.010	0.038	0.044		-	0.060	-	
Average of Ages over 10 Years	0.146	0.020	0.060	0.077	0.097	0.018	0.023	0.061		0.059	0.058	0.15	
	-	(46%)	-	-	-	(46%)	-	-		(50%)	(78%)	(50%)	
	0.140	0.020	0.063	0.065	0.104	0.015	0.039	0.052		0.042	0.058	0.17	
	-	(55%)	-	-	-	(38%)	-	-		(9%)	(45%)	(9%)	

*The numbers in the upper row of each age group pertain to the apex area, those in the lower line to the aortic region.

Table 8 illustrates the normal values of sound durations and intervals as compiled by Luisada; our own data are presented on page 364, original text.

Calo (1951) speaks about the presence of an auxiliary sound which appears within the ventricular musculature and, to a lesser extent, within the valve apparatus of the heart after the period of the rapid filling of the ventricles during the phase of "elastic ventricular action." Calo named this sound "the V sound." This author asserts that with careful examination it can often be detected about 0.08-0.16 sec. after the III sound. During normal auscultation this sound is ignored due to the very low frequency of its oscillations. Snellen and Hartman (1961) assert there are six distinct sounds: I, II, III, IV, the systolic sound of expulsion, and the tone produced by the opening of the atrioventricular valves.

Rodbards (1957) describes an unusual approach to the analysis of the sounds and noises of the heart. He proposes that these acoustical phenomena are formed by separate transitory sounds which are fused together into a single sound as a result of the longer duration of their low frequency components. The author proposes a filtration of the low frequency oscillation in order to obtain the individual, high frequency components of short duration. Each of these components would then correspond to the opening or shutting of particular valves. /290

Interesting results are obtained by means of intracardiac phonocardiography. With the use of this method Yamakawa et al. (1954) discovered the presence of turbulent motions within the heart chambers and vessels. These sounds cannot be auscultated at the surface of the chest because of the damping effect produced by the various partitions of the heart and of the vessels. Using this method Deitz, Wallace and Brown (1958) demonstrated that sound intensity on the left side of the heart is greater than on the right side. Lewis et al. (1957) discovered the following phenomena: that the I sound exhibits its maximum intensity in the ventricle; the II sound is most intensive in the region of the pulmonary artery; the III sound is discernible only in certain cases; the IV sound is very audible in the area of auricles and disappears with the flickering of the auricles; there is always a noise within the pulmonary artery, but it is not discernible on the surface of the chest. While performing intracardiac phonocardiography it must be considered that the motion of the catheter within the heart and in particular in the vicinity of the heart valves may produce sound phenomena. (Eldridge and Hultgren, 1958). Such noises are easily recognizable because of their randomness and non-conformity with the phases of the cardiac cycle.

IV. PATHOLOGICAL CHANGES IN THE PHONOCARDIOGRAM

With pathological conditions of the heart there appear various changes in the amplitude, frequency and duration of sounds. These sounds can split themselves and appear as new sounds and supplementary sounds. Various other noise phenomena also develop. It must be taken into consideration that the entire nature of a phonocardiogram may be changed due to various physiologi-

cal factors. We have already discussed the effect produced by the act of respiration. Quite often the character of the acoustical phenomena changes with alteration of the position of the body. Thus, it is well known that with the subject positioned on his left side, the acoustical complex of a mitral defect is easily detectable (Fig. 106). When the subject is in a sitting position, tilted forward and slightly to the left, there is marked accentuation of the diastolic noise produced by defective functioning of the aorta.

When one of our patients was in a recumbent position, we were able to register a melodic systolic noise. When the same patient was in a standing position, the typical melody of mitral stenosis was heard. Physical stress increases cardiac sounds and may produce the phenomena of noise. Every acceleration and deceleration of the heart rhythm becomes a source of appearance or disappearance of certain acoustical phenomena. /291

V. PHONOCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE SYNTHETIC ELECTROCARDIOLOGIC COMPLEX

1. Physical Structure of Heart Sounds

If there is an increase in duration of sounds I and II, this increase must be regarded as a noise phenomenon. An accentuated tone is determined by an increase in amplitude of a group of oscillations or of all the oscillations of the sound. L. M. Fitileva (1962) suggests that the intensity of the I sound can be judged by the magnitude of the amplitude of its basic part at the heart apex as compared with that of the amplitude of the II sound. Thus, the I sound is abnormally strong if its amplitude is two times larger than that of the II sound; if its amplitude is above that value, the I sound becomes a knocking sound. The I sound is considered weakened when its amplitude is equal to or less than that of the II sound. The intensified I sound occurs with mitral stenosis (Figs. 105, 106, 104), tachycardia, anemia, and other conditions where there is an insufficient filling of the ventricle and the stroke of the atrioventricular valves is increased. Intensification of the II sound is observed in conditions which are associated with hypertension in the aorta or the pulmonary artery (Fig. 115).

In certain pathological conditions, especially in the presence of mitral stenosis, a retardation of the I sound appears, i.e., an increase of the Q-I interval on the phonocardiogram (Figs. 105-106). The mechanism of this phenomenon is not yet exactly known, but it is known that it depends on a combination of electrical and mechanical factors, (Weissler, Leonard and Warren, 1958). It may be asserted that in this case there is a prolongation of the tensile phase of ventricular systole and in particular of the period of transformation.

The splitting of sounds is of considerable significance. In practice we encounter quite frequently the splitting of the I sound at the heart apex, or at the V point. The parts of the split sound have an analogous aspect and the interval between them is usually 0.04-0.06 sec. Such splitting

can also be observed in a healthy individual. It is slightly increased during inhalation. In pathological cases it is often observed with a bundle branch block due to retarded closing of the atrioventricular valves of that half of the heart where interventricular transmission is disturbed.

The splitting of the II sound is more readily observable in the area of the heart base. It develops when the semilunar valves of the aorta and the pulmonary artery do not close at the same instant; this leads to an increase of that short interval (0.02-0.03 sec) which is normally observable between the instants of the closure of these valves. Potin has observed the physiological splitting of the II sound in children. This splitting is cyclic, in accordance with the phases of respiration and is increased during inhalation and disappears (or considerably decreases) during exhalation. This physiological splitting is explained by the fact that during the inhalation, expulsion of blood from the right ventricle is somewhat prolonged because an inspiratory increase of the blood flow into the right ventricle begins at that time. As a result there occurs a retardation of the closure of the pulmonary artery valve. Two mechanisms operate during the exhalation phase. In the first place, there occurs a decreased blood flow into the right ventricle. As a result of an early closure of the valve of the pulmonary artery, the pulmonary component of the II sound approaches the value of the aortic component; in the second place, as under these conditions there is an increase in volume of the left ventricle, the closure of the aortic valve is retarded and the value of the aortic component of the II sound approaches that of the pulmonary component and, sometimes blends with it. The physiological splitting of the II sound may reach the value of 0.07-0.10 sec. Pathological splitting of the II sound (Fig. 107a) is observed when there is retardation in valve closure in the pulmonary artery or in the aorta caused by prolongation of right ventricular systole or by retardation in the excitatory process of one of the ventricles. A splitting of the II sound occurs at the expense of retardation in the pulmonary component whenever pathological conditions exist which cause an increase of pressure within the pulmonary circulation, and which lead to systolic or diastolic hypertension of the right ventricle, or to retardation of its contraction. Thus, a phonocardiogram taken at the region of the pulmonary artery shows a split II sound with an increased second (pulmonary) component whenever there exist such conditions as: mitral stenosis, defective interauricular or interventricular septa, prior (essential) pulmonary hypertension or a pulmonary syndrome of the heart. A similar picture is observed with stenosis of the pulmonary artery. In this case, however, the second component of the split sound is of small amplitude. With aortic stenosis, optimal splitting of the II sound occurs to the right of the second intercostal region and is due to retardation of the aortic component which is also of a very small amplitude. In the case of mitral valve insufficiency, as well as with a defective interventricular septum, the splitting of the II sound occurs at the expense of early closure of the aortic valve as a result of shortening the left ventricular systole. With a bundle branch block, the splitting of the II sound proceeds at the expense of retardation of closure of the semilunar valves of the vessel originating at the affected ventricle.

In certain cases the so-called paradoxical splitting of the II sound appears (Gray 1956; Grishman and Bleifer, 1960); namely, when the interval between the two portions of the split sound decreases during inhalation, and increases during exhalation. In such cases the aortic component of the II /293 sound does not precede the pulmonary component, but follows it. Such a condition is common when there is a left bundle branch block caused by retardation in the onset of left ventricular systole, as well as in certain cases of aortic stenosis and patent ductus arteriosus due to prolongation of left ventricular and shortening of right ventricular systole.

2. Additional Sounds

During the phase of heart contraction certain additional sounds may be heard together with the four normal sounds. Some of these additional sounds may be of considerable importance. While characterizing these sounds we use their classification by Wolferth and Margolies (1957) as a base.

A. The tone or the click of the opening of the mitral valve (Figs. 105-107) Normally, the opening of the mitral valve is not accompanied by new sounds. When, however, there is an increase of pressure in the left auricle or in the case of nonelastic dense valves, this heart mechanism causes a sharp click. This tone was described by past authors, but its detailed characteristics were obtained by means of phonocardiography. This high frequency tone is regarded as a pathognomonic symptom of mitral stenosis, and the time interval between its appearance and the II sound varies between wide limits depending on the degree of stenosis (see below). According to intracardiac phonocardiography (Deitz, Wallace, and Brown, 1958), the sound is generated within the left auricle and propagates into the left ventricle. With severe destructive alterations or calcification of the mitral valve, this sound either disappears or its intensity is very much decreased. In certain cases of tricuspidal stenosis there is registration of a click at the opening of this valve. This tone is characterized by comparatively low amplitude oscillations and is of short duration (Fig. 109) (L. M. Fitileva, 1961).

B. Clicks of expulsion (systolic clicks) These sounds were described in 1937 by Lian and Welti under the name of "claquement protosystolique" (protosystolic click). Later, Wolferth and Margolies designated them as the clicks of the opening of the semilunar valves. Clicks of blood expulsion during the early period of the systole are similar to the pattern of the splitting of the I sound because they overlap the second half of the I sound. The second component, however, is of a different configuration and is intensified. These early systolic clicks partially reflect the later phase of the systolic tension of the ventricles and, essentially, the whole phase of blood expulsion. While making the analysis of the phonocardiograms of 809 patients, Minhas and Gasul (1959) discovered an early systolic click in 135 cases; mid or late systolic clicks were found in only 11 cases. These /294 authors think that early systolic clicks represent pathological phenomena. Other types of clicks are observed in healthy individuals. It must be noted that all mid and late systolic sounds have, basically, an extracardiac ori-

gin and appear as a result of heart motion within the mediastinum and also because of friction with the adjacent tissues and pleural or pericardial membranes. These click variations have no constant localization in the different cycles of heart activity.

Early systolic sounds consist of high frequency oscillations and, according to Leatham and Vogelpoel, (1954) originate as a result of those oscillations which appear during the expulsion of the blood into the dilated aorta or into the pulmonary artery during the initial period of the expulsion phase. Optimum observations of aortic clicking are obtained in the area of the heart apex, probably because of the thrust of the heart apex against the chest. In rarer cases they also appear in the area to the right of the second intercostal and then propagate into all heart areas. These clicks are encountered with the coarctation, aneurism, and dilation of the aorta, with its stenosis or regurgitation, Fallot's tetrad, etc. Pulmonary expulsion clicks are encountered more often than aortic ones. Their optimal observation is in the area of the pulmonary artery. They are not heard in the region of the heart's apex and, in contrast to aortic sounds, their intensity varies in different respiratory phases. Maximum intensity occurs during the phase of exhalation, and they appear quite frequently in connection with pulmonary hypertension of differing causal factors. According to a series of authors, they may also be caused by moderate stenosis of the pulmonary artery.

C. Auricular sounds. Under this heading we consider the oscillation which begins after wave P, just before the beginning of the QRS complex of the electrocardiogram. It is divided from the II sound by an interval greater than 0.23 sec. Defining the sounds in this manner Kincaid-Smith and Barlow (1959) subdivide an auricular sound into two components: audible and inaudible. Only the second component is present in a healthy individual. Wolferth and Margolies (1957) distinguish the early auricular sound in the area of the heart apex, 0.08-0.14 sec. after the beginning of wave P of the phonocardiogram, and the later type which appears in the third or fourth intercostal areas, 0.24-0.30 sec. after the start of wave P. The auricular sound appears with hypertension (Fig. 117), coronary insufficiency, atrioventricular block, etc. We, however, agree with Levine and Harvey (1949) that, essentially, no auricular sound can be detected by means of ordinary auscultation.

D. Friction noise of the pericardium. This noise consists of high frequency oscillations. Its frequency reaches 600 oscillations per second. The noise is frequently composed of three parts (Harvey, 1961). The first, or presystolic part is connected with the systole of the auricles (provided there is a sinusoidal rhythm); the second, or systolic part, appears during the systole of the ventricles, while the third, or diastolic part, is formed during the early or middle periods of diastole at the time of rapid blood flow into the ventricles. Differentiation of any simultaneously occurring noises is extremely difficult. /295

E. Other sounds. Noises produced by air oscillations within the stomach are very seldom encountered; they appear as a massive oscillation in the

epigastric region and originate due to energetic contractions of the heart when there is air and liquid in the stomach. With a pneumothorax, a pericardial knock occurs, reminding one of broad systolic oscillations; sometimes a diastolic component also appears, but is not continuous. It may occur during different systolic phases and is most audible at the apex of the heart. Due to these peculiarities, this sound can be easily distinguished from systolic clicks (Johnston, 1938). Sounds due to mediastinal emphysema are similar to pericardial sounds; their occurrence is very rare.

3. Three-Beat Rhythm

Introduction of phonocardiographic studies in clinical practice led to alteration of the whole series of theoretical and practical concepts underlying methods of heart auscultation. From this point of view a considerable change occurred regarding the concept of three-beat and four-beat (galloping) rhythm described by the founders of contemporary cardiology. Such rhythm is no longer regarded as the only phenomenon of auscultation and it is no longer possible to ascribe grave prognostic value to it as was done before.

Trigeminal, or three-beat, rhythm is encountered on phonocardiograms taken from both healthy and sick individuals although it is not always apparent by auscultative means. In each case it is necessary to distinguish physiological from pathological three-beat rhythms. We find the term, "galloping rhythm" is applicable to three-beat rhythm only when it is observed in a pathological heart. After the study of this topic, Luisada (1953, 1957) came to the conclusion that any diastolic sound having a frequency of 30 or more oscillations per second and/or an amplitude exceeding $\frac{2}{3}$ of the amplitude of the I sound at the heart apex must be considered as pathologic. According to Ongley (1960), an important condition for recognition of the rhythm as "galloping" is acceleration of the heart-beat rhythm to not less than 100 contractions per minute.

A series of attempts at classification of diverse three-beat rhythms is described in the literature on phonocardiography. This question is rather complicated because in every individual case of three-beat rhythm, the origin may be different. Frost (1949) distinguished two types of gallop: (the auricular gallop and the gallop of a rapid blood flow). Evans (1951) recognized three types of the trigeminal rhythm: /296

- three-beat rhythm as a result of the appearance of the III sound;
- three-beat rhythm as a result of the appearance of auricular sound due to retardation of atrioventricular transmission;
- three-beat rhythm as a result of the appearance of additional sounds during the late period of systole.

Luisada and Aravanis (1957) offered a broader and better classification from a clinical standpoint. They distinguished three following modes of three-beat rhythms.

A. Three-beat rhythm occurring as a result of an increase in diastolic sound. - Diastolic Gallop. This mode can be subdivided into three types (Luisada, 1953; Contro, 1957; Harvey and Stapleton, 1958):

The Ventricular Type (Fig. 108). This type is caused by an increase in volume or frequency of the III sound. It is most audible in the area of the heart apex at the V point, about 0.12-0.20 sec. after the II sound. It is often encountered in young, healthy people; in adults it appears when a condition of tachycardia or ventricular hypertension exists. This rhythm occurs quite frequently in connection with defects of the interventricular septum and with insufficiency of the mitral valve. It may also occur as a result of insufficiency of the left or, rarer, right ventricle. This type may also be labelled as protodiastolic gallop.

The Auricular type (Fig. 117). This type is caused by an increase in volume or frequency of the IV sound. It is more audible in the area of the heart apex or in the third or fourth intercostal space either to the right or to the left of the sternum. It appears 0.08-0.14 sec. after the beginning of wave P. It can also be observed in healthy individuals. In pathological cases, it accompanies illness which leads to the heightening of interauricular pressure and to development of hypertrophy, hypertension, aortic defects, overloading of the ventricles, etc. This type can be labelled as presystolic gallop.

The Summary Type is caused by summation of the two diastolic sounds, III and IV. It is most observable in the area of the heart apex at the V point approximately 0.12-0.20 sec. after the II sound, and consists of low frequency oscillations. It occurs in the middle of diastole under conditions of tachycardia caused by severe shortening of the interval between the period of rapid auricular blood flow and auricular contraction.

B. Three-beat rhythm caused by the addition of a systolic sound (Systolic click). This category contains those cases where there is an appearance of systolic sounds or clicks of blood expulsions (for details see page 293 orig. test). Accordingly, it is possible to distinguish several such types of rhythm depending on the period of systole during which the sound occurs; most frequently there occurs a mid-systolic gallop. /297 Wolferth and Margolies (1957) propose to distinguish two types of systolic gallop: the aortic and the summit (apex) gallop, depending on the optimal point of auscultation. It must be noted that the existence of systolic gallop is not generally accepted. For example, Lian (1961) is against acknowledging the existence of such a gallop. We think that from the physical point of view and on the basis of phonocardiographic diagrams, the existence of this type of systolic click rhythm must be admitted, although systolic gallop is of no significance from a pathological standpoint.

C. Three-beat rhythm due to the increased intensity of the tone produced by opening of the mitral valve (Mitral click). This symptom appears with, and is characteristic of, a mitral stenosis. It reminds one of a sound such as "Obraztsov's quail", consisting of a clicking I sound and a split II sound. It may occasionally be assumed that the rhythm of the "quail sound" is

sound is due to this type of gallop rhythm.

4. Murmurs

Heart murmurs are produced by both hydraulic and anatomic factors. The main cause of murmur formation is an acceleration of blood flow (frequency) through constricted passages and the consequent appearance of turbulences. (L. A. Oganessian, 1940, 1943). S. F. Oleynik gives the following characteristic of murmurs. "A cardiac murmur originates as a result of oscillations of blood particles and vibration of various structural formations. A murmur consists of the turbulent and tissue sounds. The turbulent component only appears behind the place of constriction, while the tissue component occurs within the constriction and behind it."

Phonocardiograms register murmurs as groups of similar or dissimilar oscillations with respect to their amplitudes and frequencies. The pattern of these oscillations depends on the character of the murmur. The intensity of a murmur is determined by the amplitude of its vibrations; the frequency characteristic is determined by measuring the frequency of oscillations and the timbre depends on the periodicity and the regularity of oscillations. The chronological order of a murmur is determined by comparing the localization of these vibrations with the phase of heart contraction. In determining the temporal relationship of a murmur we use the electrocardiogram. There we find the first (I) and second (II) heart sounds and consequently the phases of systole and diastole. Cowen and Parnum (1949) are of the opinion, however, that electrocardiography cannot serve as a reliable criterion for determining the phase characteristic of a murmur because these diagrams do not define the relationship between the electrical and the mechanical events of heart contractions. For practical purposes, Evans /298 (1957) suggests drawing a vertical line parallel to the registered electrocardiogram from the end of wave S to the phonocardiographic curve (on the second channel). All murmurs which immediately precede this line have an auricular origin, and those which occur directly after this line coincide with the early period of the systole.

With the use of phonocardiography it is possible to determine the locality - the epicenter - of murmur formation, because, as shown by Feruglio (1959), a murmur originates within that chamber or that vessel which receives the hemodynamically altered blood flow. In practice this center is determined by finding the place of optimal audibility of the registered murmur. It must be noted, however, that the point of optimal audibility of a murmur on the chest surface does not always correspond to the true locality of formation of this murmur. Thus, in the case of insufficiency of the mitral valve, the murmur originates within the left auricle, but it is more audible in the area of the heart apex; also, with insufficiency of the aortic valve, the murmur occurs within the left ventricle, but it is more audible in the area of the aorta, and with aortic stenosis the best area for auscultation frequently is not the aortic area, but rather that of the pulmonary artery, or even the heart apex.

Consideration must be also given to the shape or the silhouette of the

group of oscillations pertaining to this or another murmur (Fig. 104A). Under certain conditions the study of only the silhouette of the noise oscillations may be of great help in the correct interpretation of the diagram. Phonocardiograms display very well types of crescendo and decrescendo. In the first instance the amplitude of an oscillation is gradually increasing while in the second, it is as gradually decreasing (Figs. 106, 107, 115). The murmurs with initial crescendo oscillations which subsequently become decrescendo oscillations, appear characteristic of a rhombus, or a diamond, or finally, of a pulsation wave (Figs. 111, 112). The silhouette of individual murmurs resembles a concertina: during the entire extent of the murmur there are alterations in its intensity and groups of identical amplitudes appear (Fig. 114). A relatively characteristic pattern is obtained for musical tones. These are loud murmurs, and they reflect very well the hemodynamic condition (McKusick, 1957).

All available murmurs can be divided into two general groups: systolic and diastolic. Their characteristics can be given according to the following physical factors: time of appearance, duration, intensity, pitch, timbre and shape.

In the literature there is a description of a whole series of methods for classifying diverse murmurs. In our work we prefer the classification of Luisada (1953, 1957) and, especially, that of Leatham (1953, 1958) as being the most beneficial for clinical investigations. /299

According to Luisada's classification, all systolic murmurs can be divided into two large groups: those of the apex of the heart and those of its base.

General classification may be accomplished in the following manner:

1. Increase of general duration of the I-sound or that of its second part only /300
2. Systolic murmur of the decrescendo type, ending prior to the appearance of the II-sound.
3. Systolic murmur of a decrescendo type, which is rarely encountered, with insufficiency of the mitral valve and with a severe aortic stenosis and stenosis of the pulmonary artery.
4. Pansystolic murmur in the area of the heart apex caused by mitral valve insufficiency, or in the area of the heart base under the condition of severe stenosis of the aorta and of the pulmonary arterial valves.
5. Systolic murmur, of a diamond shape. It is encountered at the base of the heart and is caused by stenosis of the pulmonary artery.
6. Systolic murmur, of the concertina shape; its optimum audibility is at the middle of the auricular area.

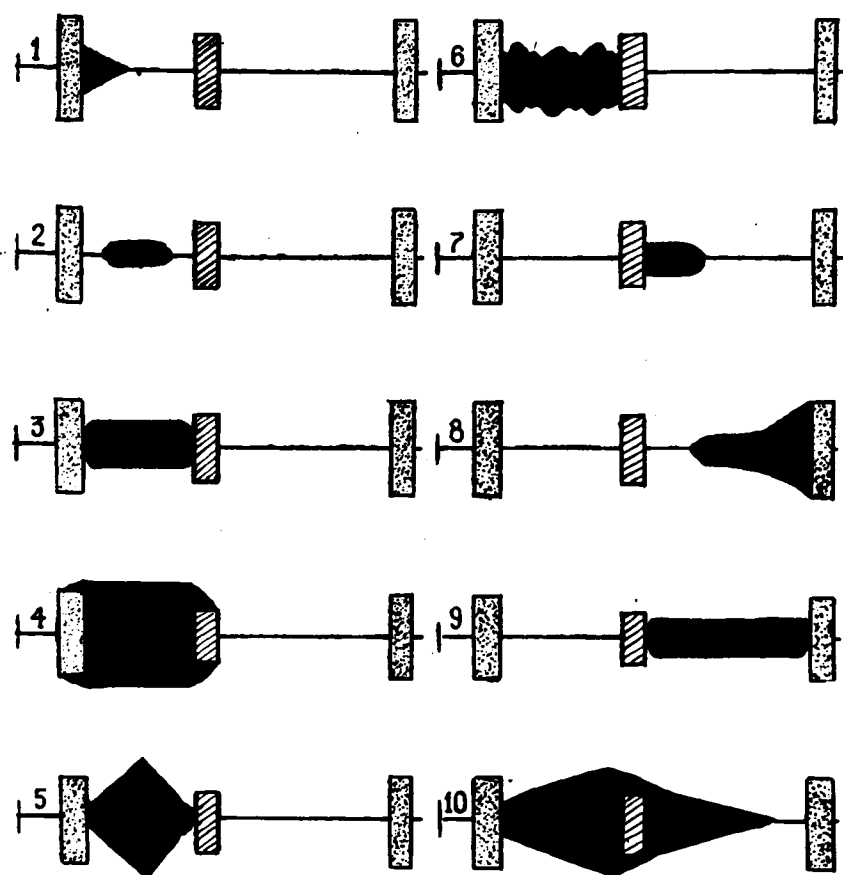


Fig. 104A. Shapes of most frequently encountered murmurs (black figures represent murmur phenomena; dotted columns represent the I sound; columns with slanting lines represent the II sound). 1. Weak, early systolic murmur of decrescendo type; 2. midsystolic murmur; 3. continuous systolic murmur which does not blend with sounds; 4. continuous intensive systolic sound blending with sounds; 5. rhomboidal systolic murmur; 6. concertina-like systolic sound; 7. protodiastolic sound; 8. mid-diastolic sound with presystolic increase of the crescendo type; 9. continuous diastolic murmur; 10. systolo-diastolic murmur of the crescendo-decrescendo type (mechanical murmur).

According to Luisada (1953, 1957), diastolic murmur occurring in the area of the heart apex appears as irregular oscillations during the early period of the diastole or during the whole diastole with an accentuation during the presystolic period. Such murmurs are observed with mitral stenosis. There are two types of diastolic murmurs at the heart base: a prolongation of the II-sound and a long diastolic murmur. These murmurs accompany insufficiencies of the valves of the aorta or of the pulmonary artery. Luisada, et al. (1957) found, that with a mitral stenosis, the diastolic murmurs are quite audible with an increase of vibratory frequency from 60 to 110 cycles per sec. For optimum auscultation of the diastolic noises occurring in connection with defects of the aorta, their vibratory frequencies must be increased from

150 to 200 cycles per second.

On the basis of the shapes of murmurs and their relation to the heart sounds, Leatham (1953, 1958), distinguishes two principal groups of systolic murmurs. The first group comprises the so-called midsystolic murmurs of expulsion which occur during the phase of blood expulsion, when the blood is forced through the constricted outlets of the aorta or the pulmonary artery. Murmurs having diamond-like, or rhombus-like shape do not blend with the I or II heart sound. They begin after the moment of the opening of the semilunar valves; their intensities increase gradually as a crescendo until the midsystole. In the second part of the systole their intensity diminishes (in decrescendo pattern), and finally, the murmur oscillations die out just before the instant of closing of the semilunar valves. These murmurs are auscultated generally at the heart base when there is a stenosis of the aorta or of the pulmonary artery (Fig. 110, 112); also in those cases when, due to an increased blood volume expelled from the ventricles, there originates a relative stenosis of the semilunar orifices (this may occur in connection with defects of the interauricular or interventricular septa, anemia, /301 pregnancy, etc.). We agree with Master, Donoso and Rosenfeld (1960) that such murmurs are audible also when there is a stenosis of the outgoing ventricular vessels. The second group of systolic murmurs comprise the so-called pansystolic regurgitational murmurs which occur during the regurgitation of the blood, or during its flow through obstacles (defects), from a chamber of higher internal pressure to a chamber whose internal pressure is lower. These murmurs extend through the whole systole and blend with the I and II sounds. During the entire systole, the intensity of these murmurs changes very little and cannot therefore, serve as a criterion for judgement regarding the degree of anatomic damage, as the intensity depends almost entirely on the speed of the blood flow. Basically, these murmurs are observed in connection with insufficiency of the mitral valve and with defects of the interventricular septum (Figs. 108, 114).

It is useful to observe the long systolic murmur of extracardiac origin which is sometimes auscultated in pregnant women, especially in the area of the heart base and which occurs due to expansion of the mammary artery. This murmur is very well described by Grant (1956). In analogy with the so-called "uterine souffle," it is called by name of "mammary souffle." It disappears when pressure is applied to the stethoscope.

According to Leatham (1953, 1958), diastolic murmurs are divided into three groups. The first group comprises murmurs due to blood flow into the ventricles. To this group belong diastolic murmurs which are formed with stenosis of the atrioventricular openings, which form after the closure of the semilunar valves. Quite often these murmurs are intensified in the presystolic phase (Figs. 105, 106, 107). With a severe rheumatic endocarditis there occurs a midsystolic murmur due, probably, to the inflammation of the valves. With an increased blood flow through the atrioventricular valves, there occurs a short diastolic murmur during the phase of rapid blood inflow. This murmur is produced by the relative stenosis of these orifices generated under these conditions.

The second group comprises murmurs caused by contraction of the auricles. A typical example of such murmurs is the presystolic murmur occurring with mitral stenosis (Figs. 106, 107). The third group contains the regurgitational murmurs which are caused by organic insufficiency of the aortic (Fig. 111) or pulmonary valves, and also by the relative insufficiency of their orifices (with aneurism, hypertonia). These murmurs are characterized by a high frequency.

There also exist the so-called continuous murmurs which are auscultated during the whole cycle of the cardiac mechanism. Such long murmurs occur with an patent ductus arteriosus (Fig. 113), arterio-venal fistula and with certain types of coarctation of the aorta. In fact, the already described "mammary souffle" and the venous murmur (rumble) are such murmurs. Lian, Minot and Welti (1951), describe these continuous murmurs in the region of the right second intercostal along the line of the sternum. These murmurs occur with /302 constriction of the superior vena cava due to aortitis or mediastinitis; they may occur in the region of the right scapula with the inflammatory constriction of one of the pulmonary veins.

Separate consideration must be given to the inorganic, functional, or as certain authors call them, innocent murmurs. In general, these murmurs originate as a result of an abnormal increase of the velocity of blood flow or due to an increase in blood volume; basically they are low intensity, systolic murmurs, most often registered in the area of the pulmonary artery. They do not extend over the entire systole but occur in decrescendo form at the beginning of the systole and at the midsystole. In general, functional murmurs are high frequency oscillations and are of a very unstable character, changing very rapidly with changes of body position. R. D. Dibner (1958) notices that in a series of cases, their amplitude decreases after some physical loading. After an examination of children, Fogel (1960) found functional systolic murmur in 66.7% of the cases. Fogel proposes elucidation of the following types: vibratory by-sternum-precordial, blowing pulmonary, and cardiac-respiratory. From a clinical standpoint, a better classification was made by Evans (1947), who, after an examination of 330 people with observable functional murmurs, subdivided them into the following five categories:

1. Murmur in a recumbent position. This is a low-intensity murmur disappearing with deep inhalation. Frequently it is encountered in young people.
2. Murmur in a standing position. This is a blowing, somewhat coarse murmur, also encountered in young people.
3. A loud murmur which reminds one of the murmur occurring in connection with mitral valve insufficiency.
4. Parasternal murmur audible in the IV intercostal in the region of the left edge of the sternum.
5. Murmur occurring at the end of the systole. This is a loud murmur observable at all ages.

L. A. Antonova (1960) finds functional murmurs in young patients with hypertension. The murmurs are systolic and are most audible in the region just over the heart and large vessels including the abdominal aorta. The author also finds diastolic murmurs of the esophagus. These murmurs are not discernable by ordinary auscultation. The author thinks that the appearance of these murmurs is due to increased blood flow, which in turn is due to intensified cardiac activity during conditions of low peripheral resistance.

In practice, one may encounter functional diastolic murmurs. A century ago, Flint described a functional diastolic murmur occurring in connection with insufficiency of the aortic valve. L. M. Fitileva (1962) records functional murmurs, frequently appearing at the middle or at the end of the diastole and propagating over large areas. Z. K. Chkhaidze (1961) describes /303 presystolic and diastolic murmurs of functional origin occurring in patients with general atherosclerosis, nephritis and inflammation of the internal organs. In all cases, autopsy failed to reveal any alteration of the cardiac valve apparatus.

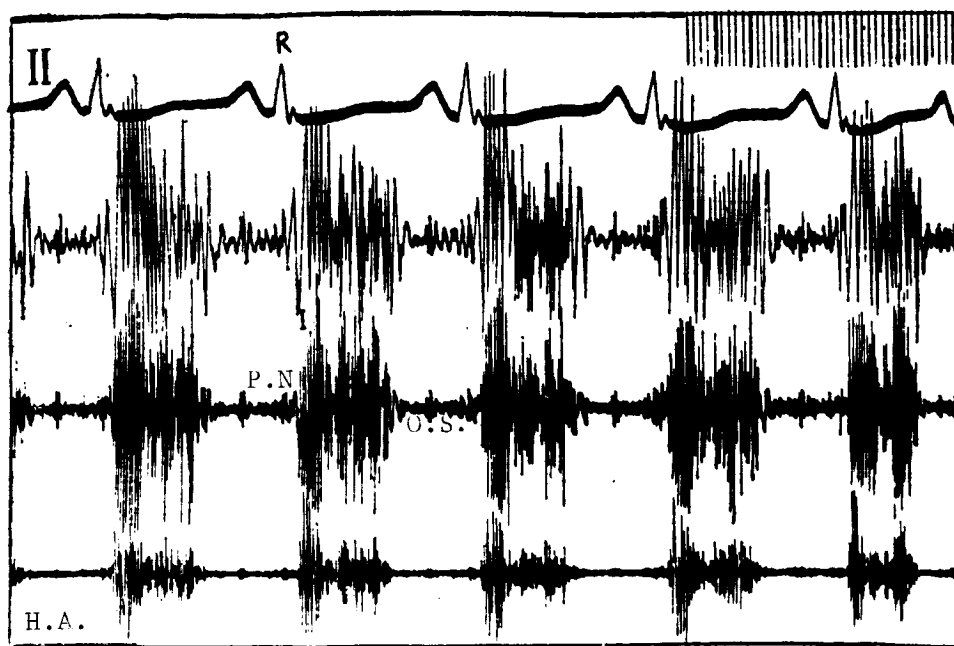
Various functional tests are used for recognition and exposure of functional noises, such as: alteration of body position, physical loading, certain medications, etc. Wells (1954) suggests registering the phonocardiogram at rest as well as with an application of the Valsalva test. Of great interest in this connection is an attempt by Crevasse (1959) to use a vessel-contracting agent for the diagnosis of functional murmurs. Thus, with the use of such an agent as merphentermine for example, the character of functional murmurs is usually not altered, while the systolic murmur which is due to insufficiency of the mitral or aortic valves is increased as a result of an increase in aortic pressure, while the murmur which is due to insufficiency of the pulmonary artery valve remains unchanged because pressure within the pulmonary circulation remains unchanged.

5. Phonocardiographic Data for Certain Diseases of the Cardiovascular System

A. Mitral stenosis (Figs. 105, 106, 107). In the presence of mitral stenosis, the phonocardiographic pattern is characterized by a series of variations discussed in 1862 by Duroziez, who identified the patterns of this defect with an expression "ffouttatarou." In this case the I sound, particularly its mitral component, becomes intensified, flapping and is somewhat retarded. The frequency of the I sound is also changed reaching a value of 713 cps; at maximum intensity, the frequencies reach a value of 360 cps (G. I. Kassirskiy, 1960). The II sound is intensified and is often split, especially in the area of the pulmonary artery.

Very characteristic is an appearance of a click - the opening snap - of the mitral valve (OS). Many authors mention this phenomenon (I. V. Savchenkov, 1959; V. I. Maslyuk, 1959; T. G. Didebulidze, 1961; Dack et al. 1960, etc.). This murmur is quite audible at the heart apex; sometimes, however, it is not detectable at the heart base. This symptom is pathognomic for mitral stenosis, and several authors think that when it is absent, the

diagnosis of mitral stenosis is doubtful. In our practice, there were cases when mitral stenosis was recognized on the phonocardiogram only by the presence of the snap-click of this valve. We must consider, however, that in practice, this sound sometimes cannot be detected because of diastolic noises, especially in the protodiastolic phase. Besides, it can also be absent when there is considerable sclerosis of the mitral valve. This high frequency sound appears because, with the opening of the mitral valve, the blood flow from the left auricle (where the internal pressure is high) suddenly meets an obstacle.



/304

Figure 105a¹

We agree with L. M. Fitileva (1962) that there are no compelling reasons to favor another explanation, other than the fact that this sound appears as a result of a retardation and intensification of the third part of the II sound.

It is necessary to distinguish the opening click from the split II sound.^{/307} In most cases this is possible, because with splitting, the two sound components usually resemble each other and the interval between them does not

¹Symbols appearing in this and subsequent figures: H.A.- apex of the heart; L.S., area of the heart apex with body positioned on the left side; 0 - zero point, A - aortic area; P.A. pulmonary artery area; III-IV, intercostals to the left of the sternum; T - the tricuspid valve area; S. N. - systolic noise; D.N. - diastolic noise; P.N. - presystolic noise; O.S. - click of opening snap of the mitral valve. Above the curve there is instruction relating to the electrocardiogram tracing.

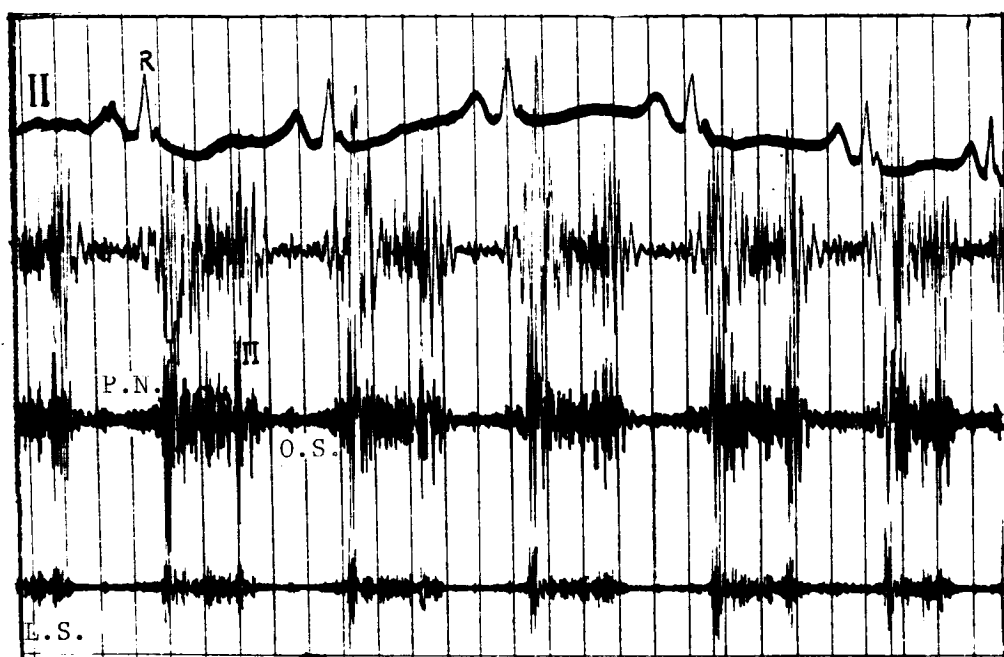


Figure 105b

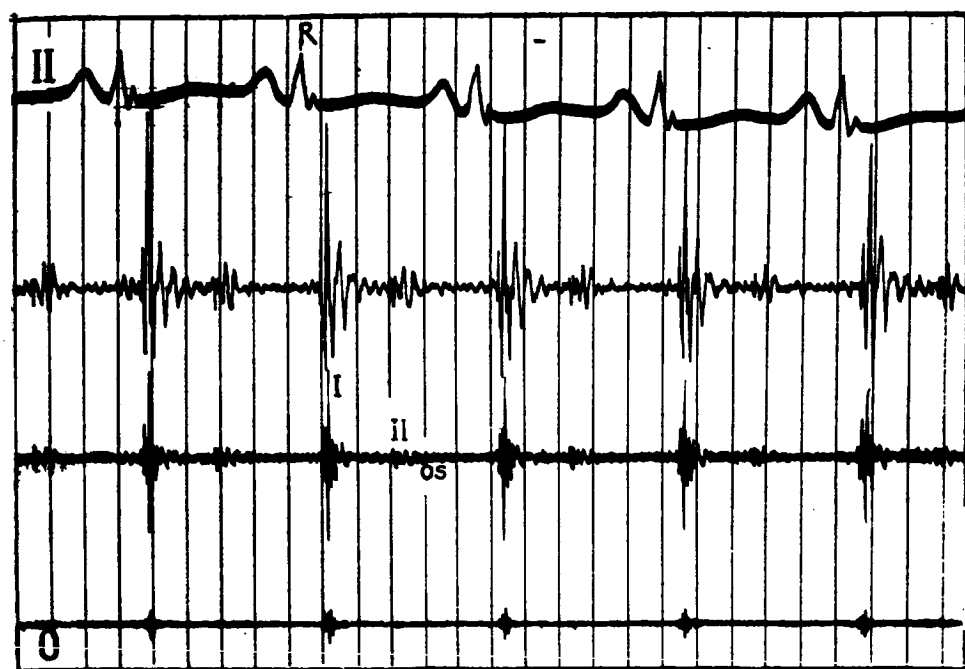


Figure 105c

Fig. 105. Mitral stenosis. Phonocardiograms of patient B. O., female, age 31. Diagnosis: Mitral defect with predominant stenosis. (a) at the heart apex Sound I is intensified; there is a continuous systolic noise; diastolic noise with a crescendo type presystolic increase; the click of opening snap of the mitral valve; $Q-I = 0.07$ sec. (b) while lying on the left side, the I sound increases; the systolic side weakens somewhat. (c) at the zero point the I sound is increased; noise phenomena disappear.

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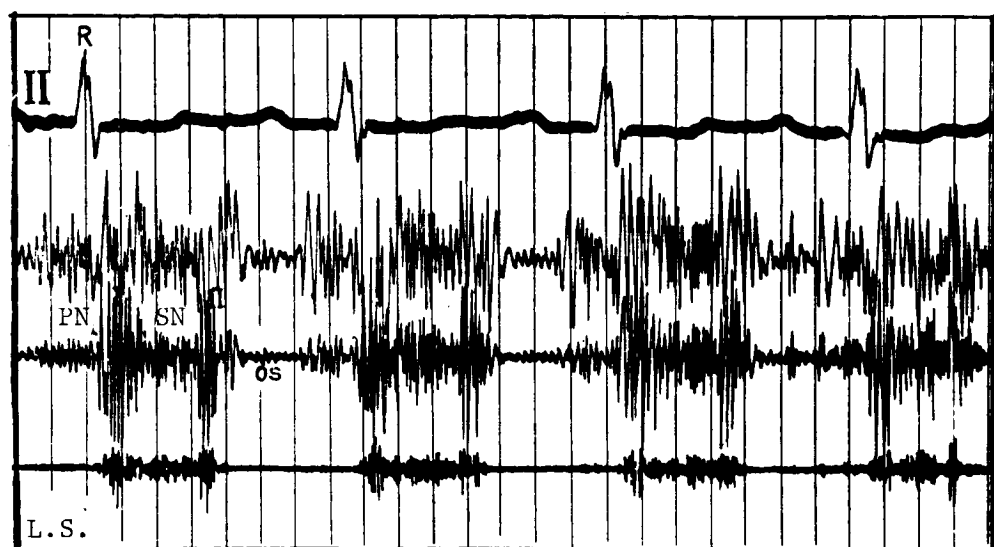


Figure 106a

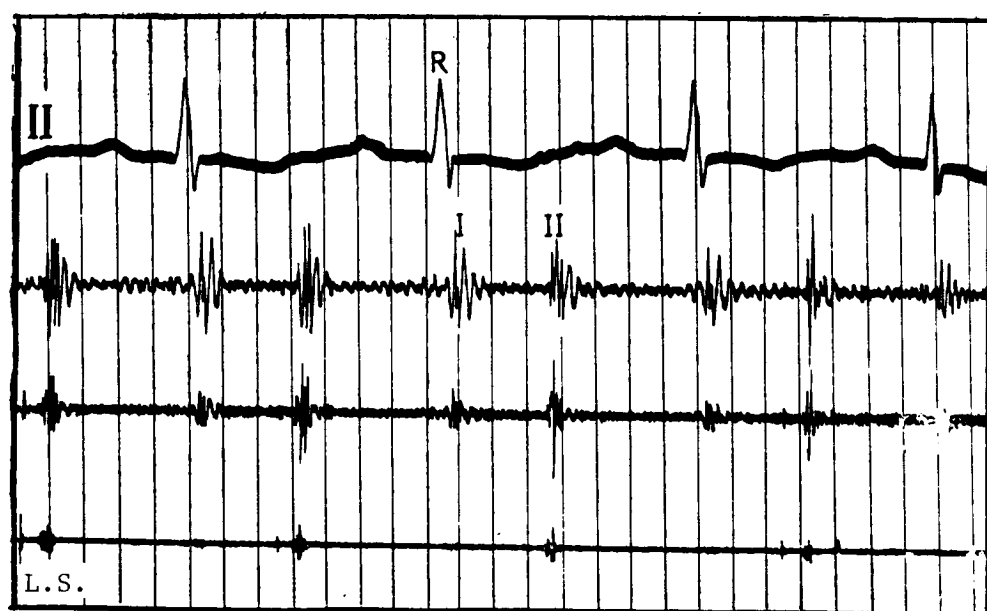


Figure 106b

Fig. 106. Mitral Stenosis. Phonocardiogram of patient J. A., female, age 25. Diagnosis: Mitral defect with predominance of stenosis. (a) when in lying position on the left side, sounds I and II at the heart apex are increased; continuous systolic sound (disappearing at the zero point); crescendo type presystolic sound; click of opening snap of mitral valve, Q-I = 0.07 sec. (b) one month after a mitral commissurotomy, the I sound is weakened; noises and mitral click disappeared. Q-I = 0.06 sec.

exceed 0.06-0.07 seconds, while the interval between the II sound and the click is usually longer than this time interval. Also the shape of the

click sound differs from that of the II sound.

The III sound cannot be auscultated because the blood flow into the left ventricle is severely diminished. Only seldom can it be detected on the cardiogram even when there is good myocardial tone and a moderate degree of stenosis, but with the discovery of a real ventricular gallop, one must suspect the presence of other cardiac defects (Contro, 1957). It is not difficult to distinguish the III sound from the opening click, because it is of low frequency, weak, and appears 0.12-0.13 seconds after the II sound.

In the presystolic phase, a low frequency diastolic noise occurs which increases in a crescendo manner. Presystolic noise appears in interval P-R i.e., during the contraction of the auricles (Evans, 1947, Nylin and Biörck, 1947). It may also be detected, however, during fibrillation and flutter of the auricles. In the latter case, the presystolic noise appears only with rapid contractions of the ventricles. When, due to an early appearance of sound I, the two noises blend together with a decrease in the number of ventricular contractions, there appears a diastolic noise which stops before the advent of the I sound. T. S. Gal'perina (1959) observed a coarse noise appearing sometimes at the beginning, sometimes at the middle and, sometimes at the end of the diastole, thus appearing as a "wandering" noise. With the advent of the decompensation of the right side of the heart and with a development of the relative insufficiency of the tricuspid valve, the presystolic noise frequently disappears altogether, probably due to the decrease of pressure within the pulmonary circulatory system and within the left auricle.

It must be noted that with mitral stenosis, there frequently appears at the heart apex a systolic noise which in certain cases is not accompanied by a distinct diastolic noise. In such cases, if the phonocardiogram is registered from the zero point, the systolic noise frequently disappears entirely, and a characteristic acoustical pattern of mitral stenosis is observed. G. G. Gel'shteyn and L. M. Fitileva, (1959) think that the blending of this noise in the axillary area with a knocking I sound and with presystolic noise of varying intensity is a symptom of a severe contraction of the left venal orifice. Quite correctly, these authors connect the appearance of systolic noise with the development of functional stenosis of the pulmonary artery, or with a relative insufficiency of the tricuspid valve, which occurs whenever there is severe dilation of the right ventricle. Consideration must also be given to those sclerotic cicatricial alterations on /308 the folds of the mitral valve which, while causing the insufficiency of the valve, also cause the regurgitation. In these cases, the left-side position for taking the phonocardiograms intensifies the systolic noise. A clever interpretation of the origin of this noise is given by I. I. Savchenkov (1960) who, on the basis of X-ray endoauscultation, concludes that systolic noise accompanied by a pure mitral stenosis is of an intraventricular origin. It appears as a result of the vibratory oscillations of the sinewy and papillary muscles accelerating blood inflow and outflow in the left ventricle.

Two other phonocardiographic symptoms of mitral stenosis are also of considerable importance: Interval Q-I (interval from the start of QRS to the beginning of the maximum oscillation of the II sound) and the II-OS interval (from the start of maximum II sound oscillation to the start of oscillations of the mitral click). According to many authors, the duration of these intervals determine the degree of development of the stenosis. It is directly proportional to the length of interval Q-I and inversely proportional to that of II-OS. Gallavardin and Delahaye, (1957) think that an increase of interval Q-I is a clear symptom of stenosis. Wells (1954, 1957) submits a method of determining the degree of the stenosis by means of characteristics Q-I, II-OS and the electrocardiographic interval R-R of the preceding heartbeat cycle. He thinks that if $(Q-I) - (II-OS) > 1$, there is, in all probability, a high gradient of pressure and a severe stenosis. If, however, this difference is less than -1.5, there is a low pressure gradient and the area of the mitral orifice is larger than 1×1 cm. We find that variations of these intervals only have relative significance in the determining of the degree of the stenosis and are not of a constant value. These symptoms, especially interval II-OS, give only a very general conception of the degree of stenosis. The reason for this is that an increase of interval Q-I occurs also in a series of other heart diseases, and the click of the opening snap of the mitral valve frequently blends with the diastolic noise and cannot be exposed. In addition to this, Ye. A. Popova (1961) finds that an increase of interval Q-I does not always appear with mitral stenosis, and interval II-OS determines only the presence of the stenosis but not its degree. We think that the character itself, or rather the duration of the diastolic noise, may serve as a very good indicator of the degree of stenosis. With a moderate degree of stenosis, this noise is registered only in the middle of the diastole but with severe stenosis it is prolonged and a presystolic increase of its intensity occurs, because there is then a high gradient of pressure during the entire period of blood influx. In favor of this opinion are the data obtained with an intracardiac study of the sonic phenomena accompanying mitral stenosis (Moskowitz, et al. 1960). One must remember however, that the intensity of the diastolic noise alone is not a sufficient criterion for determining the degree of stenosis. Equally significant in the evaluation of the stenosis is the intensity of the I sound. There is a definite relation between the stenosis and this increase of sound intensity. /310

It must be noted that in practice, such typical phonocardiographic patterns are not always obtainable. In certain cases diastolic noise is very weak and a pattern of systolic noise prevails. In other cases the diastolic noise is entirely absent. We must underline the fact that in certain rare cases there is a complete absence of the noise pattern on the phonocardiogram. (Mute patterns, according to L. M. Fitileva, 1962). Then, a presence of mitral stenosis can be detected only by means of other criteria, especially that of the click of the opening snap.

After surgery (Figs. 106b and 107b), the character of the auscultation patterns can be considerably changed, although the observed alterations are usually not related directly to the clinical condition of the patient. Comberiati and Collicelli (1954), also, do not find a regular connection

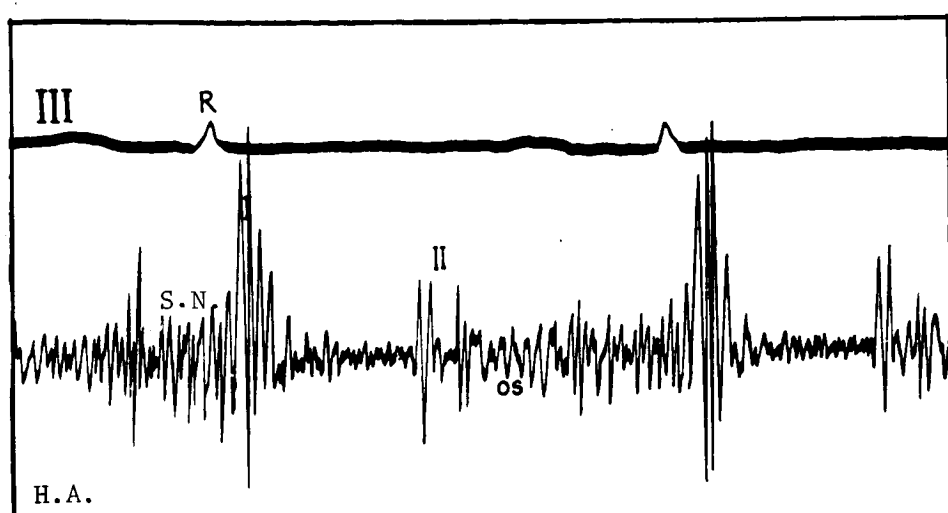


Figure 107a

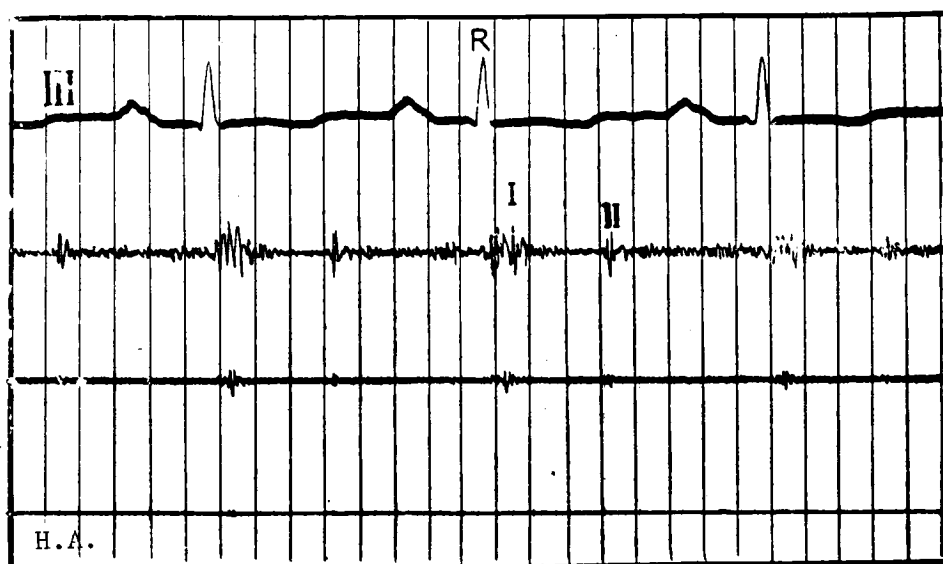


Figure 107b

Fig. 107. Mitral stenosis. Phonocardiogram of patient A. I., female, age 22. Diagnosis: Mitral defect with predominance of stenosis. (a) on the heart apex the I sound is increased and the II sound is split; there is a snap-click upon opening of the mitral valve; weak early systolic noise; diastolic noise with crescendo-type presystolic intensification; $Q-I = 0.05$ sec. (b) 10 days after mitral commissurotomy: the I sound is not increased; the II sound is not split; amplitude of diastolic noise is considerably decreased; presystolic intensification disappears; $Q-I = 0.04$. (Intensification of the II sound on pulmonary artery disappears.)

between the clinical improvement and the pattern of the acoustical phenomena. G. I. Kassirskiy(1960), however, observed a certain parallel between the

diminution of the frequency of the I sound with the clinical effect of the surgery. Evaluating distant follow-up results of a mitral commissurotomy, Ye. N. Meshalkin, I. N. Meshalkin and Ya. S. Vaynbaum (1961) observed considerable alterations in the auscultation pattern. We find that a considerable diminution of the diastolic and, especially, of the presystolic noise, diminution or disappearance of the systolic noise, diminution of the Q-I interval, diminution of the amplitude of the I sound at the apex and that of the II sound at the pulmonary artery are sufficiently reliable criteria to judge the effectiveness of the mitral commissurotomy. In rare cases, the diastolic noise remains unchanged or is even increased, evidently, as a result of the formation of coarse alterations in the area of the mitral valve, and, as shown by E. N. Dembovskaya (1962), of an increase of the blood flow through the mitral orifice. According to I. A. Kassirskiy and G. I. Kassirskiy (1961), in 25% of patients operated upon the diastolic noise remains unchanged.

B. Insufficiency of the mitral valve (Fig. 108). The I sound usually remains unchanged, or there occurs a slight decrease of its amplitude. The II sound is sometimes increased, but more often, becomes split as a result of an early closure of the aortic valve. As is noted by Perloff and Harvey (1958), the interval between the two split components of this sound is, rather wide and constant. The III sound is almost always present; it is especially intensified in the region of the heart apex and is a result of a large blood flow into the left ventricle. A high frequency regurgitative systolic noise is observed which endures throughout the whole systole, /311 often increases at the end. In principle, it is possible to agree with V. I. Maslyuk, I. I. Sivkov and N. L. Yastrebtsovoy (1961), that there is a connection between the audibility of the systolic noise and the degree of mitral insufficiency. This noise is sometimes of a decrescendo type. Quite frequently, a diastolic noise of short duration can be detected at the beginning or in the middle of the diastolic period, especially in the area of the heart apex. According to Hubbard, Donn and Neis, (1959), this noise is caused by a low atrioventricular pressure gradient during the phase of blood flow into the ventricles. These authors state that in the presence of a sinusoidal rhythm and with increased atrioventricular transmission, there may occur a presystolic gallop and presystolic noise. Bleifer, et al. (1960), point out the occurrence of the mid-diastolic noise following the III sound (the Kari Kumbz noise), can be explained by the increased volume of blood passing through the mitral orifice. Accompanying the protodiastolic noise, K. P. Buteyko and L. M. Yagafarov (1961) detect, sometimes, an increase of the I noise on the heart apex.

It must be observed that with insufficiency of the mitral valve, the click of its opening snap is not observed, although, Deshmukh, Uricchio and Likoff (1958) have detected this sound in a series of patients with pure mitral regurgitation proven during surgery or during autopsy.

C. Defects of the tricuspid valve (Fig. 109). With insufficiency of the tricuspid valve the I sound is not weakened because the intensity of this sound does not mainly depend on the closure of the tricuspid valve but on /312 that of the mitral valve. There appears a high frequency regurgitative

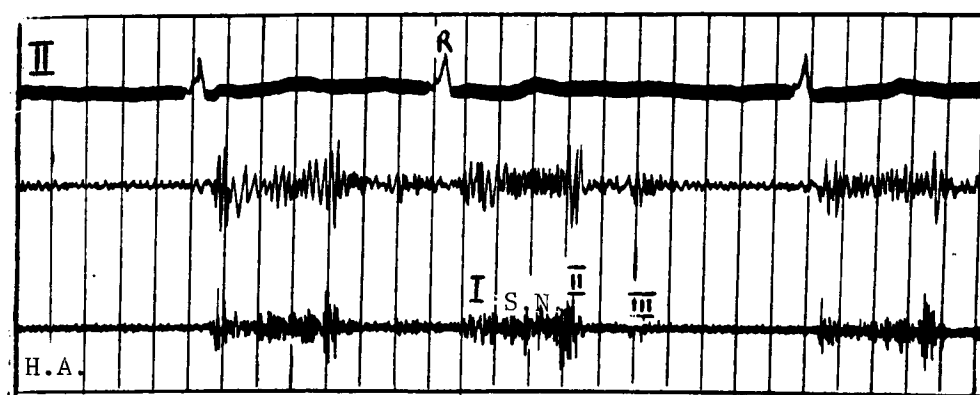


Fig. 108. Insufficiency of the mitral valve. Phonocardiogram of patient O. V., male, age 34. Diagnosis: Mitral defect with a predominance of insufficiency; at the heart apex the I sound is periodically weakened; the III sound and the regurgitative systolic noise can also be distinguished.

systolic sound which is most audible in the area of the xiphoid process the sound is transmitted quite well to the right, lower portion of the chest and is slightly increased during inhalation. This noise does become weaker towards the region of the apex and therefore an inverse pattern is not entirely excluded. The sound intensity depends on the degree of the insufficiency. In general, however, this noise is weaker than that occurring with mitral insufficiency. According to I. V. Shaban (1961) an inverse interdependence may also be encountered.

Diagnosis of tricuspid stenosis is difficult. It is accomplished by mid- or presystolic noise and by the click of its opening snap. The latter differs from the mitral click by its shorter duration and a weaker intensity. These acoustical phenomena are increased by holding the breath at the phase of deep inhalation (the Rivero-Corvallo system, according to L. M. Fitileva, 1962). This is probably due to the increased venous blood flow into the right auricle.

Isolated tricuspid defects are very rare. More often they are observed in conjunction with defects of other valves, and in particular with those of the mitral valve.

D. Aortic stenosis (Fig. 110). There is an observable diminution in amplitude of the II sound and sometimes this sound is split into two components. Sound IV is frequently detected.

There is an observable systolic noise which is most audible by auscultation in the region of the aorta. Sometimes it is even more audible to the left of the sternum, especially in the area of the pulmonary artery; it can also be detected at the heart apex. The systolic noise is also well auscultated on the vessels, especially the carotid. This noise starts after the end of the I sound, its intensity is gradually increased up to the middle part, or to the second half of the systole and then it decreases and

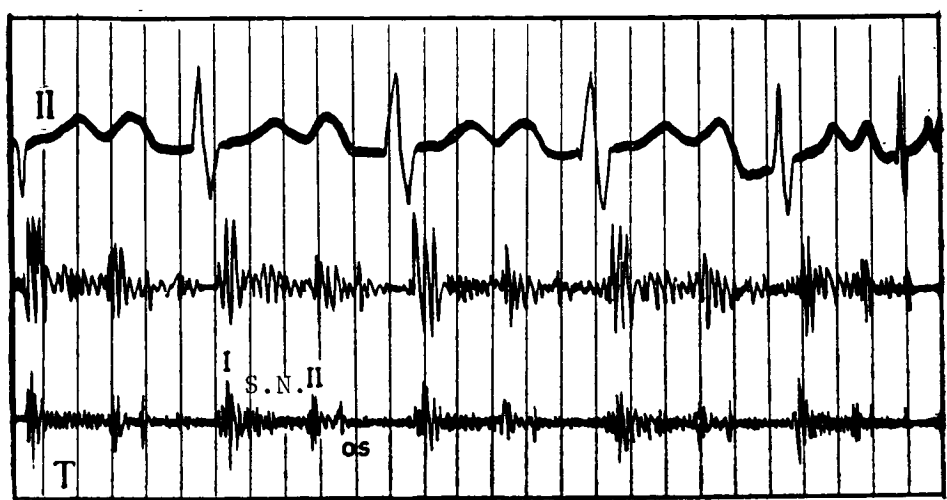


Fig. 109. Tricuspid defect. Phonocardiogram of patient E. R., male, age 23. Diagnosis: Mitral-tricuspid heart defect. Sound I is increased in the area of the tricuspid valve (T); there is a click at the opening snap of this valve; there is a systolic noise and very weak diastolic noise.

disappears almost entirely with the advent of the II sound. In other words, the midsystolic noise of expulsion is of a rhomboidal shape and differs considerably from systolic noise caused by insufficiency of the mitral valve. The latter is evenly distributed during the entire systole and increases somewhat only at its end. According to localization of the maxima of these noises, it is possible to distinguish the early and the late systolic noises (Aravanis and Luisiada, 1957); many observations by A. L. Mikaelyan (1962) show a predominance of the late sound. According to intracardiac phonocardiography there is a quiet period between this noise and the first component of the I sound. This period corresponds to the phase of the isometric contraction of the left ventricle. (Moscovitz, et al. 1960). The location of the apex of the rhomboidal noise may, to a certain extent, serve as a criterion for determining the degree of aortic stenosis: if the apex lies closer to the I sound, the stenosis is mild but if the apex lies in the middle or closer to the II sound, there is a severe aortic stenosis. It must be stressed that the intensity of sound is not a criterion for determining the degree of the stenosis. Successful surgical correction of the aortic stenosis may lead to a complete disappearance of this noise.

E. Insufficiency of the aortic valve (Fig. 111). Amplitude of the I sound is very low. There is a frequent click upon opening of the aortic valve. High frequency diastolic noise is observed. Its oscillations appear directly after the II sound. Toward the end of the diastole, these oscillations gradually slow down and frequently disappear in the presystole. This noise may be of various intensities. It begins in the early diastole, after the closure of the aortic valve, because it is exactly during this period that a strong gradient of pressure forms between the aorta and the left ventricle. In addition to this, it lasts throughout the entire diastole (the pandiastolic noise) because this gradient is of a relatively

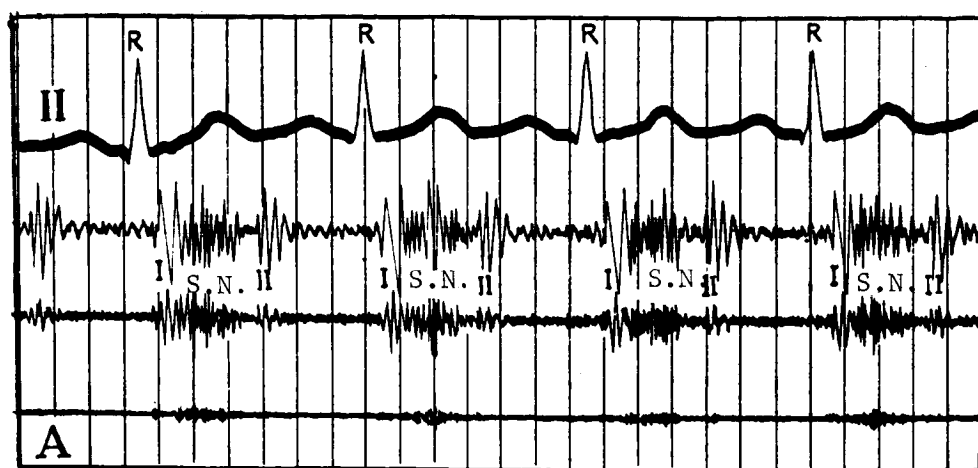


Figure 110a

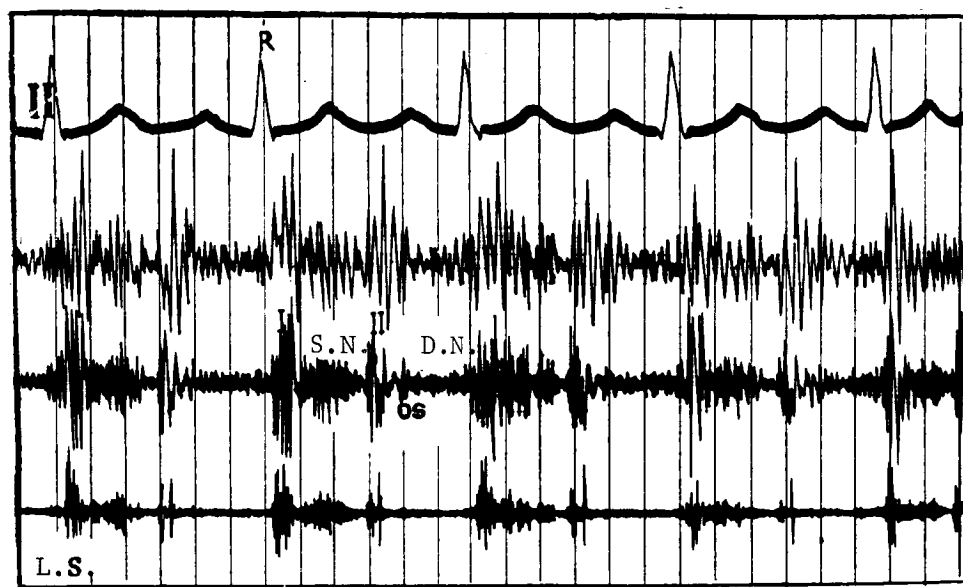


Figure 110b

Fig. 110. Aortic stenosis. Phonocardiogram of patient P. Z., female, age 34. Diagnosis: Combined aortic-mitral stenosis (a) on the aorta, there is a weak II sound; rhomboidal systolic noise ending before the advent of the II sound. (b) When the patient is on his left side, sounds I and II are intensified; there is a click at the opening snap of the mitral valve; rhomboidal systolic noise from the aorta, although here it is more intense than at the aorta; diastolic noise.

high level during the entire diastole. Due to this mechanism, the noise sometimes assumes an initial crescendo form which later changes to that of decrescendo. Frequently Flint's presystolic murmur also appears as a

result of the relative stenosing of the mitral orifice.

F. Coarctation of the aorta. Usually the I and II sounds are increased. A systolic noise is registered in the right side region of the heart base and also on the back of the patient. The noise may be of a spindle shape. Wells, Rappaport and Sprague (1949) detect a systolic and an early diastolic noise of a decrescendo type on the back. These noise phenomena are not so frequent/315 at the precordial area and are of a lower intensity. According to P. N. Mazayev, et al. (1957), roentgenologic endoauscultation permits exposure of the extent and form of the aortic coarctation, velocity of the blood flow, oblique blood circulation, elasticity of the vessel walls and other details.

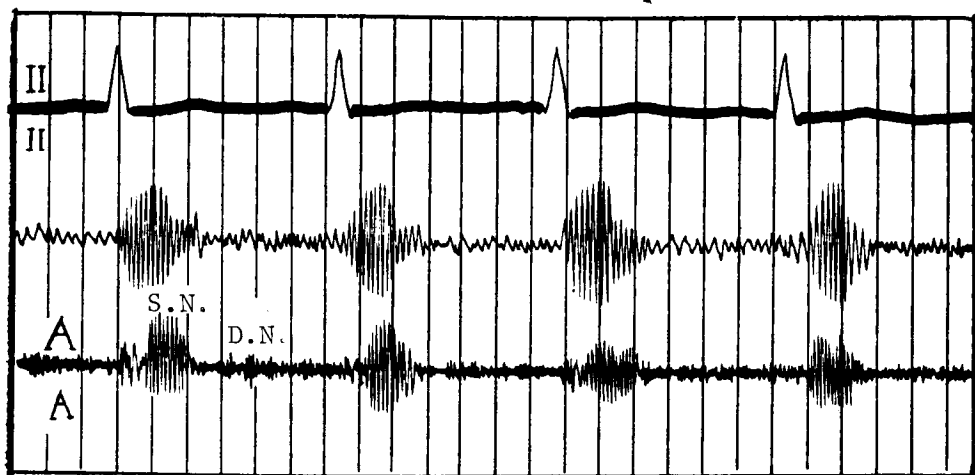


Fig. 111. Insufficiency of the aortic valve. Phonocardiogram of patient S. E., female, age 26 years. Diagnosis: Mitral-aortic heart defect. In the region of the aorta, the sounds are weakened; there is a rhomboidal systolic noise and a continuous diastolic noise.

G. Stenosis of the pulmonary artery (Fig. 112). The I-sound is almost unaltered or is accentuated in the area of the third and fourth intercostals to the left of the sternum as a result of a very strong contraction of the hypertrophic muscles of the right ventricle and an increase of the pulmonary component of the I sound. A splitting of the II sound occurs as a result of retarded closure of the pulmonary artery valve; sometimes the IV sound is registered and frequently there appears an early click of blood expulsion. There is a rhomboidal-shaped systolic noise with its apex at the middle of the systole. This noise starts after the I sound and ends prior to the advent of the II sound, although, with a severe stenosis, it overlaps the II sound and its apex approaches this sound. It must be stressed that the amplitude does not indicate the degree of stenosis. Leatham and Weitzman, (1957), Benchimol and Dimond (1958) find a linear relationship between the degree of stenosis (determined by the aortopulmonary interval) and the systolic pressure within the right ventricle. The first two of these authors as well as Crevasse and Logue (1958), on the basis of catheterization and surgery, distinguish

three groups of pulmonary artery stenosis. The first group is that of slight isolated stenosis. The electrocardiogram remains unchanged, systolic pressure within the right ventricle is lower than 40 mm Hg; there is an early click /316 of expulsion and a splitting of the II sound at the expense of the pulmonary artery component (aortopulmonary interval is 0.03 - 0.06 sec.); there is also a feeble systolic noise at the middle of the systole. The second group is that of a moderate or severe degree of stenosis. Systolic pressure in the right ventricle is equal to 50-150 mm Hg; there is a severe splitting of the II sound (aortopulmonary interval varies from 0.06 to 0.14 sec. in accordance with the intraventricular pressure). An intensive systolic noise appears overlapping the aortic component of the II sound; systolic clicks of expulsion are observed only with relatively low degrees of stenosis. The third group includes cases with stenosis of the pulmonary artery together with Fallot's tetralogy. As a result of a considerable decrease in blood flow within the pulmonary artery, the systolic noise is of short duration and of low amplitude; there is no pulmonary component of the II sound.

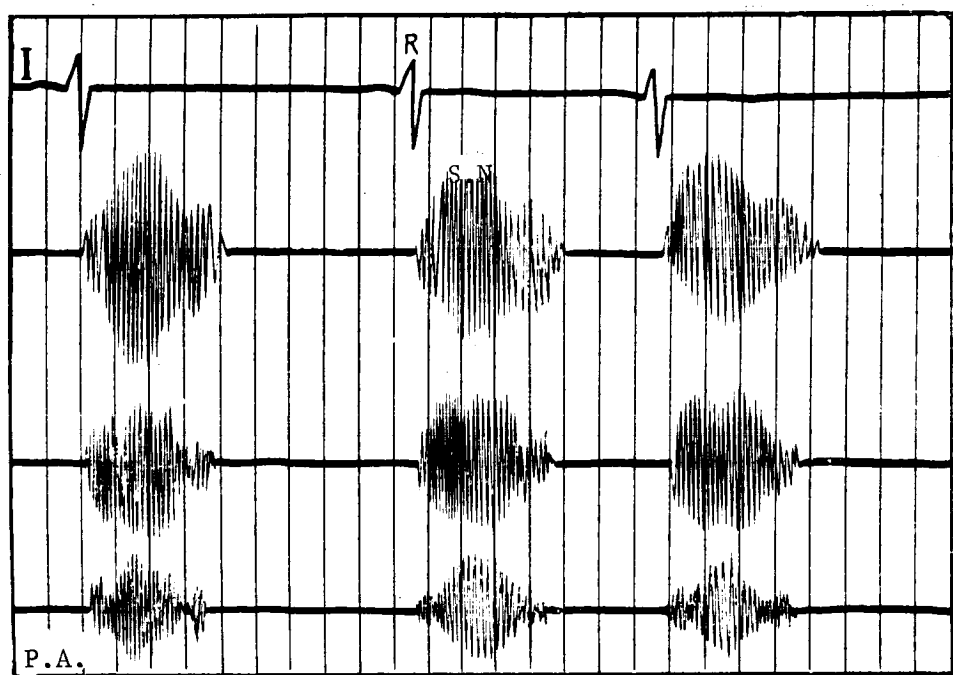


Fig. 112. Stenosis of the pulmonary artery. Phonocardiogram of patient Kh. T., age 4 years. Diagnosis: Valve stenosis of the pulmonary artery. In the area of the pulmonary artery there is an intensive rhomboidal systolic noise blended with the heart sounds.

H. Patent ductus arteriosus (Fig. 113). In the absence of other associated congenital heart defects, the I sound is usually unaltered and the II sound is often accentuated and split. The sounds are inaudible in the area of the /318

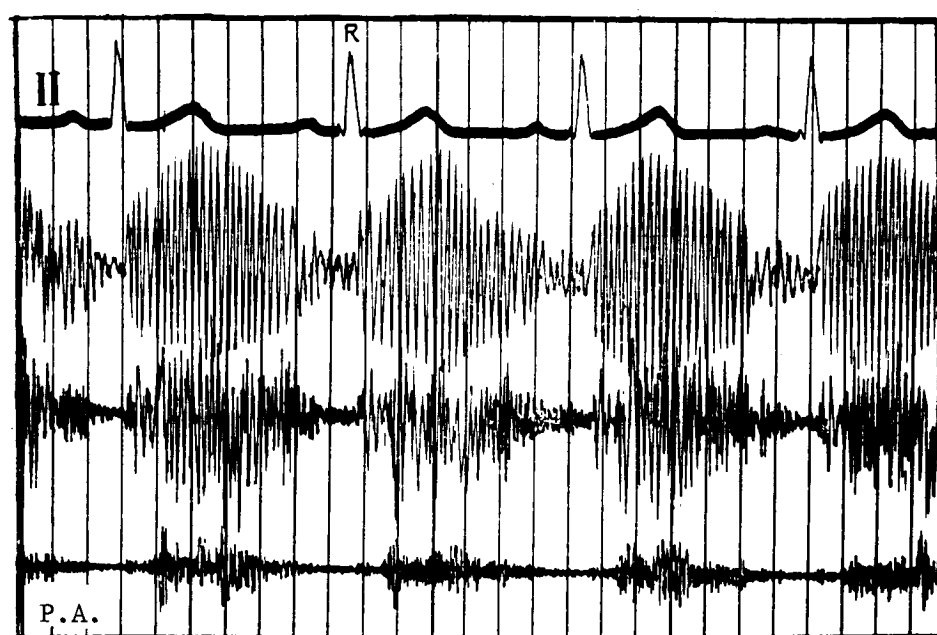


Figure 113a

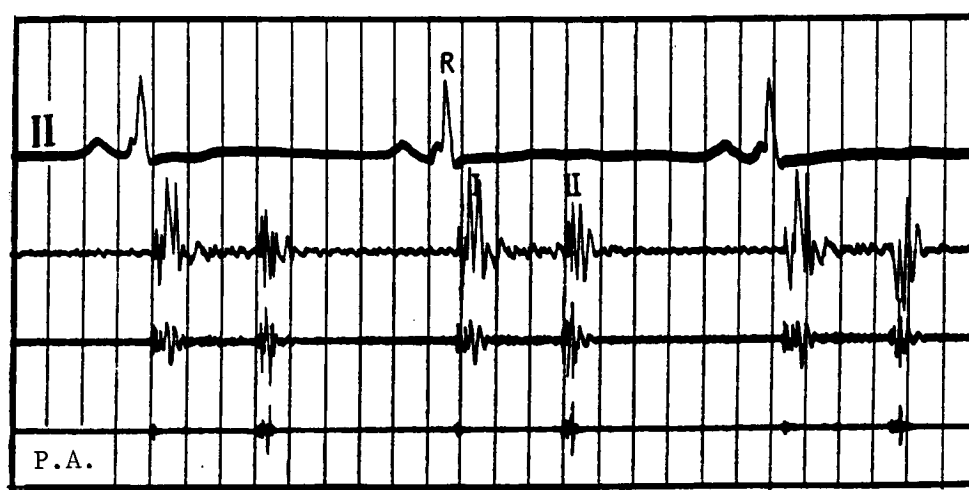


Figure 113b

Fig. 113. Patent ductus arteriosus. Phonocardiogram of patient M. V., male, age 12 years. Diagnosis: Patent ductus arteriosus. (a) On the pulmonary artery there is a continuous intensive systole-diastolic noise of crescendo-decrescendo type. The II sound is not differentiated. (b) 20 days after surgery: the noise disappears, the II sound is somewhat increased.

pulmonary artery, being masked in the background of continuous sound. The latter (mechanical sound according to Gibson) is most audible in the area of the pulmonary artery or to the left of the sternum in the area of the third and fourth intercostal. It propagates in all directions. Intensity of this sound is gradually increased at the end of the systole, but in the

phase of the diastole it assumes a decrescendo form. The increase of the noise at the end of the systole and not at its middle, with the formation of the maximum pressure difference between the two sectors, we explain by the same mechanism as do Ongley, et al. (1960): the noise appears not as a result of the blood flow through the channel, but of its passage from the narrow channel into the expanded pulmonary artery. Therefore, the "shift-to-the-left" of the noise depends on the time interval necessary for propagation of the pulsatory wave to this area. Neill and Mounsey (1958) made parallel clinical and phonocardiographic observations on postsurgical patients and conclude that this noise appears in conjunction with a slight pulmonary hypertension. This noise must be distinguished from "the mammary souffle", which may sometime assume a prolonged character (Scott and Murphy, 1958). After successful surgery this noise disappears. (Fig. 113b).

I. Interventricular septal defect. (Fig. 114). The I sound is usually not altered, or is slightly increased. The II sound is split and an intensive systolic sound is audible at the left edge of the sternum and in the area of the third and fourth intercostal. This systolic noise propagates into adjacent areas. A phonocardiogram shows a pansystolic noise blending with the I and II heart sounds; usually, maximum amplitude of oscillations is observed during the second half of the systole; the noise however, may assume a plateau form. When the defect is localized in the area of the muscular part of the interventricular septum, it can be detected only at the beginning of the systole. Systolic noise is of greater intensity, its audibility is lower, and the II sound is less split than it is with the interauricular septal defect (Brotmacher and Campbell, 1958). Intensity of the noise is considerably decreased with a decrease in the volume of the shunt from left to right and with a marked pulmonary hypertension (Hubbard, Angle and Koszewski, 1957). With severe defects, the noise considerably weakens or disappears entirely as a result of retardation of the shunt velocity, but there then appears a systolic noise of expulsion in the area of the pulmonary artery. Craige (1960) distinguishes three groups on the basis of values of pressure and blood flow within the pulmonary artery. The first group consists of cases of low pressure and blood flow; coarse pansystolic noises of crescendo-decrescendo type are registered. This noise may also be of constant intensity. A split II sound is audible, although, /319 according to Lessof (1958), with a shunt of 2 liters per minute, the II sound remains normal. Patients in the second group have a marked pulmonary hypertension and large left-to-right shunts. Loud pansystolic noises of the crescendo-decrescendo type are audible. There is also a splitting of the II sound; frequently the III sound and diastolic noises in the area of the heart apex appear as a result of rapid filling of the left ventricle. The third group comprises those cases having severe pulmonary hypertension, mixed shunts and short atypical systolic noises. Sometimes there is also a clicking noise of expulsion during the early period of the systole in the area of the pulmonary artery and an accentuation of the II sound.

J. Interauricular septal defect (Fig. 115). With this defect, all acoustical phenomena are caused by the atrial left-to-right shunt and by /320 an increase of the blood flow through the heart chambers on the right side

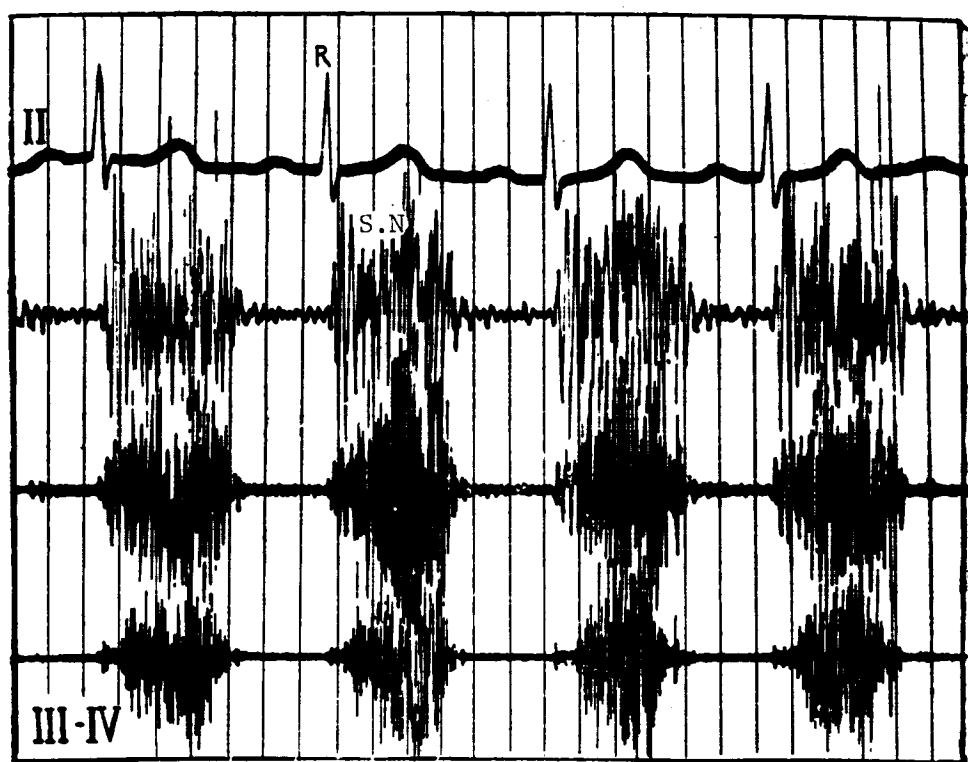


Fig. 114. Interventricular septal defect. Phonocardiogram of S. M., female, age 6 years. Diagnosis: Interventricular septal defect. To the left, in the area of the third to fourth intercostal the continuous, intensive systolic noise blends with the other noises. Isolated oscillations of noises of different frequencies and pitches. The noise joins the II sound without transition period. The II sound is split.

into the pulmonary artery. Auscultation frequently shows a splitting of the I sound. The sound is often intensified as a result of an increased blood flow through the tricuspid valve. The II sound is split due to closure retardation of the semilunar valves of the pulmonary artery. According to Boyer and Chisolm (1958), even considering the already enlarged volume of the right ventricle, respiratory variation of the splitting of the II sound disappears when inhalation is halted.

According to results obtained by Ongley (1960), a wide splitting of the II sound in the area of the pulmonary artery is frequently the first manifestation of an interauricular septal defect; this symptom is especially important in those cases where the registration of noise phenomena is very weak. A systolic noise of the expulsion type (Leatham and Grey, 1956), usually appears in the middle of the systole and is most audible in the area of the pulmonary artery. According to data obtained by means of intracardiac phonocardiography (Liu and Jacono, 1958) this noise results from an increased blood flow within the right heart chambers and the pulmonary artery, although the systolic noise sometimes does not appear on the experimental model of this defect obtained by direct heart recordings on dogs.

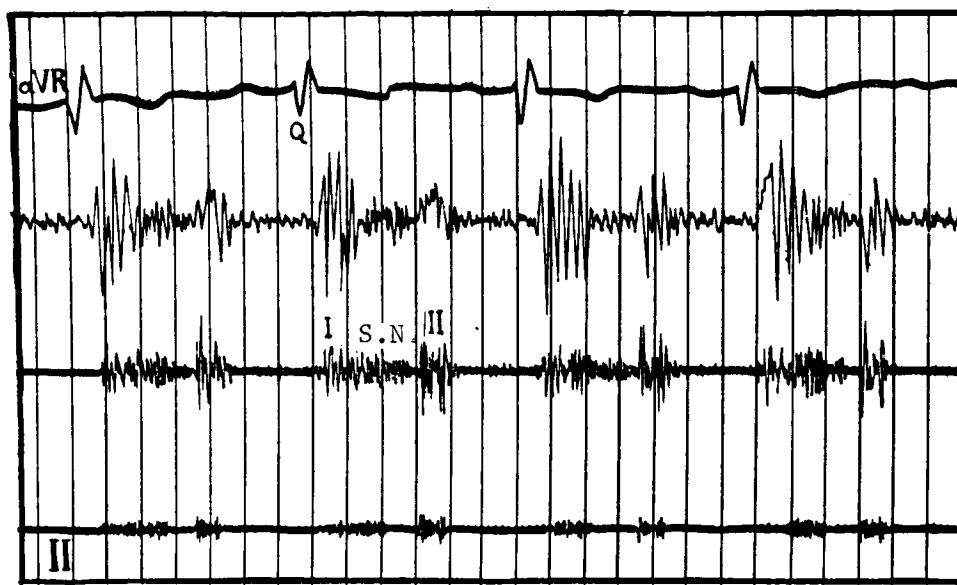


Fig. 115. Interauricular septal defect. Phonocardiogram of patient A. C., female, age 21 years. Diagnosis: Interauricular septal defect (?). To the left within the region of the second intercostal, the II sound is intensified and is split; systolic noise of medium intensity begins from the I sound; amplitude of oscillations of this noise is gradually decreased and oscillations disappear before the advent of the II sound.

A diastolic noise is audible at the middle and the end of the diastole. The early diastolic noise is audible within the left ventricle, while the short mid-diastolic noise is well registered within the right auricle and ventricle, and is formed as a result of stenosis of the tricuspidal orifice (Liu and Jacono, 1958).

K. Fallot's tetralogy (Fig. 116). The I sound is usually not changed. The II sound is weakened at the expense of considerable diminution in the amplitude of the pulmonary component. A rhomboidal systolic noise is registered in the area of the pulmonary artery and a continuous systolic noise of constant intensity throughout the whole systole is registered in the third to fourth intercostal to the left of the sternum. The first of these sounds does not blend with the I and II heart sounds, while the second does. These sonic phenomena propagate through wide areas and are not audible in the axillary area and at the upper sections of the rear surface of the epicardium. As was noticed by L. M. Fitileva (1962), these noises may sometimes be absent, firstly because of an extremely severe stenosis of the pulmonary artery and, secondly, because of a decrease or disappearance of the shunt through the interventricular septal defect.

L. Hypertensive Disease (Fig. 117). At the heart apex, there frequently appears a diminution of the amplitude of the sounds, particularly in their intermediate portions. This happens particularly during the second and third stages of the disease. In the presence of hyperfunction of the left ventricle this can be a result of a cardiosclerosis (N.Ye. Kavetskiy, A.A. Stupnitskiy,

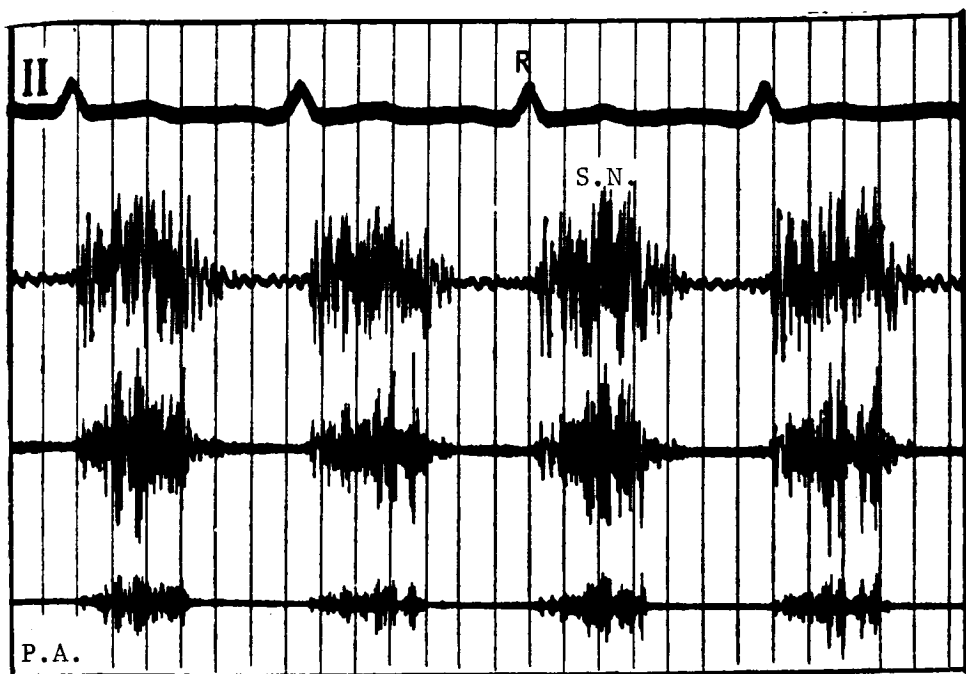
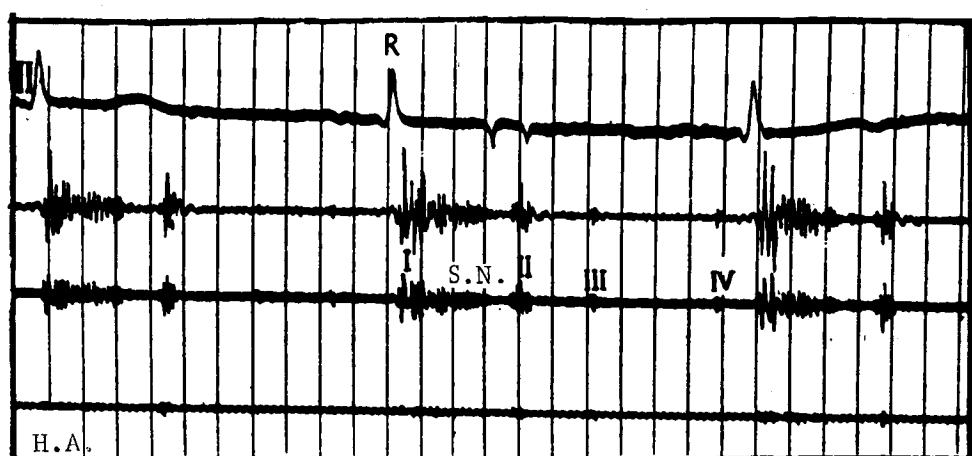


Figure 116a



Figure 116b

Fig. 116: Fallot's tetralogy. Phonocardiogram of patient V. R., female, age 17 years. Diagnosis: Fallot's tetralogy. (a) An intensive rhomboidal systolic noise blending with heart sounds. The II sound is split (?). (b) In the area of intercostal IV (to the left) amplitude of the I and II sound is increased; there is a systolic noise of medium intensity and of different configuration as compared to the noise at the pulmonary artery.



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Fig. 117. Hypertensive disease. Phonocardiogram of V. P., male, age 73 years. Diagnosis: Hypertensive disease, stage III. Atherosclerotic cardiosclerosis. On the heart apex the I sound is split; sounds III and IV are present; there is a systolic decrescendo type noise of medium intensity; interval Q-I = 0.12 sec.

1961). On the aorta, the I sound is weakened. An intensification of II sound is more often observed during the third stage of illness. In more than 50% of the cases an intensification of sound II appears in the pulmonary artery. The III sound seldom appears; more often there appears an auricular noise during the 323 third stage of illness. During the second stage there frequently appear systolic sounds. Duration of the interval "auricular sound - I sound" varies within limits of 0.03-0.06 sec. As the illness progresses there is a clear tendency toward a progressive increase of this interval. (Table 9). It is interesting to note that, with a decrease of arterial pressure, this interval decreases and, quite frequently, the three-beat rhythm of the presystolic gallop disappears entirely. Kincaid-Smith and Barlow (1959) also find that with clinical improvement, the IV sound approaches the I sound and blends with it. Although in most cases, the duration of the Q-I interval varies within normal limits, it frequently happens that there is an increase of this interval, especially during the late stages of this illness (Table 9). An increase of the Q-I interval with arterial hypertension is also shown in the data obtained by Weissler, Leonard and Warren (1958). A systolic sound of medium frequency and intensity is registered on the heart apex, aorta and pulmonary artery, in most cases during the second stage of illness. A diastolic noise is registered in only rare cases. Using a special method of registering noise phenomena, Puchner, Huston, and Hellmuth (1960) discovered a slight diastolic noise; they link it with insufficiency of the aortic valve. V. A. Triger (1951), M. A. Samoteykin (1955), T. S. Bugoslavskaya (1960) also observe the presence of diastolic noises in the area of the heart base.

Table 9

Duration of Intervals Q-I and "Auricular Sound I" at
Different Stages of Hypertensive Disease (in 140 Patients)

Duration of Interval in Seconds	Interval "Auricular sound - I. Sound"			Interval Q-I		
	First Stage	Second Stage	Third Stage	First Stage	Second Stage	Third Stage
Below 0.03	-	2	-	-	3	1
0.03 - 0.04	6	14	4	10	24	11
0.05 - 0.06	1	7	4	16	25	13
0.07 - 0.08	-	4	2	5	13	11
0.09 - 0.10	-	1	1	1	4	1
Over 0.10	-	2	2	-	-	1
Total Number of cases	7	30	13	32	70	38

VI. CONCLUSION

Although phonocardiography has a relatively long history, its actual development began only during the last two decades, because for a long time, various technical difficulties impeded its growth and inculcation into the clinic. This method is now widely used, and shows how wrong Einthoven was, when in 1907 he expressed doubt that phonocardiography would someday become a clinical method of investigation (according to Sprague, 1957).

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Phonocardiography is not only an improved method of heart auscultation, but also a great supplement to it permitting an objective study of various acoustical manifestations. Phonocardiography, however, cannot and must not replace the classical method of heart auscultation; both methods are indispensable elements of clinical examination of a patient.

Phonocardiography is of great importance in the anatomical diagnosis of various acquired or congenital heart defects. The importance of this method, particularly in synthetic electrocardiology, becomes very clear in the study of the physiology and pathophysiology of the heart cycle and the hemodynamic interpretation of heart contraction. In this respect, combined investigations with electrocardiography and arterial sphygmography play a very important role in determining the condition of the functional heart (see next chapter). The most recent information with regard to inter-relation of the dynamics of the intraventricular pressure with the genesis of cardiac sounds stresses, to an even greater extent, the hemodynamic importance of phonocardiography. Duchosal (1948) was correct when he asserted that the final word has yet to be said on cardiography, but we also agree with Py (1957) that we should not expect from it more than it can give.

CHAPTER III

COMBINED POLYCARDIOGRAPHIC INVESTIGATION: ELECTROCARDIOGRAPHY, PHONOCARDIOGRAPHY AND ARTERIAL SPHYGMOGRAPHY

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I. INTRODUCTION

In preceding chapters we presented a description of techniques and the importance of individual methods of electro- and phonocardiography. A simultaneous application of these two methods in parallel with the registration of arterial sphygmography presents the possibility of introducing ourselves into the study of a very important aspect of cardiac activity, namely, that of determining the separate phases of ventricular contraction. Such a combined investigation assumes a new significance and one must therefore make a separate examination of this problem, after having become acquainted with the technique of obtaining an arterial sphygmogram and its pattern.

II. TECHNIQUES OF SPHYGMOGRAPHY

After the initial description of the mechanical recording of the arterial pulse by Marey in 1863, this question became a subject of study by many authors. One hundred years ago, Weber made an initial experimental study of the pulsation wave (according to Porjé, 1946), and in 1905 Frank obtained an exact recording of the arterial pulse with the use of a specular capsule giving a detailed description of the pattern of its curve. In 1937 Gomez and Langevin used the piezoelectric principle for recording the arterial sphygmogram. It is possible to make aortic sphygmogram recordings (aortography had been studied in detail by Porjé in 1946) of the central pulse (that of the carotid and subclavian artery), as well as the peripheral pulse (that of the humeral, femoral and other peripheral arteries). It is also possible to record a cardiogram from the heart apex which, according to 326 Benchimol, Dimond and Carson (1961) assumes an especial importance in the study of diastolic events of the cardiac cycle. In our own work we make a recording of the carotid pulse because it is easily accomplished and the obtained curve is easily read. A linear microphone is used for this purpose (see page 252). In our practice we use the sphygmograph of type SG-01 serving as an attachment to a five channel electrocardiograph. A pulse receiver (volume pick-up unit), made in the shape of a funnel and connected by means of a hard rubber tube to the sphygmograph, is placed within the region of the anatomic projection of the carotid sinus where the carotid may be best palpated. The sphygmograph is connected to the recording apparatus.

N. N. Savitskiy (1956) shows that the curve of the central pulse (Fig. 118a) begins with a steep ascent: initial wave (1) ends at first with a smooth, then with a steep descent (3); after a few slight indentations and a small ascent (4), the curve makes a smooth descent, interrupted once by a

small ascent (5). Toward the start of the next pulse wave, there is a pre-systolic wave (6). Bramwell and King (1942) distinguish three separate waves on a normal sphygmogram: the percussion wave appearing when pulse percussion reaches the location of the sphygmographic pick-up unit on the artery, the influx wave which is a summation of the forward-moving wave with the waves reflected from the periphery, and the dicrotic wave which is formed in early diastole, and which is due to the repetitive forward thrust of blood following its backward flow toward the closed semilunar valves.

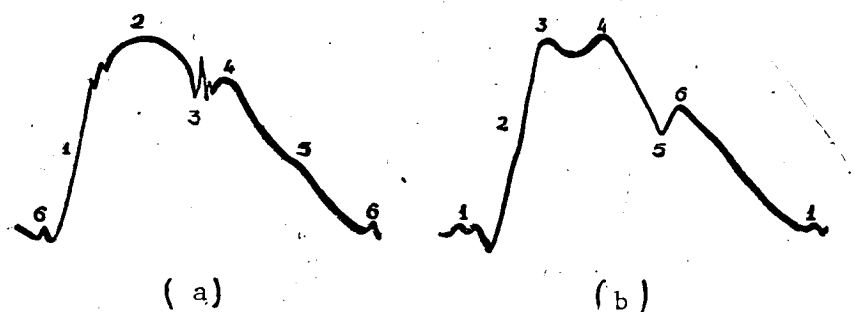


Fig. 118. Curve of the central pulse. (a) according to N. N. Savitskiy; (b) according to Luisada (explanation in the text).

Luisada (1953) gives the following description of the central pulse details (Fig. 118b):

1. During the presystolic period and during the phase of ventricular tension, there appear slight oscillations (one or two) which are produced by the auricular contraction and by the protrusion of the folds of the aortic valve. The latter is the result of the increased intraventricular pressure.

2. The percussion wave begins with a steep ascent of the pulse curve /327 (the anacrotic slope), upon which a certain kind of compression (the anacrotic compression) is noticeable. There is an anacrotic ascent which is caused by the ejection of blood (ej) from the left ventricle into the aorta. On the phonocardiogram it coincides with the large oscillations of the I sound, which correspond to the opening of the aortic valve. The anacrotic compression appears as a result of the fact that the velocity of ejection is somewhat less at the beginning of the ejection phase than it is in its later parts.

3. The apex of the percussion wave is formed at the midsystole. At this time almost one half of the percussion blood volume is already ejected from the left ventricle, and more blood leaves the aorta and enters its branches than flowed into it. After the apex there appears a mild compression.

4. During the second half of the systole, there appears a rounded influx

wave which is caused by the summation of the basic wave with peripheral waves. After this a descent of the curve begins (the catacrotic slope).

5. Upon closing of the aortic valve, an incision appears. During this period the ventricle is in the phase of diastolic relaxation and in the aorta there occurs a sudden drop of pressure.

6. Following closure of the aortic valve, the blood flow is toward the periphery, thus generating the dicrotic wave.

7. The curve's descent is gradual and toward the start of the next pulse percussion, the initial level is reached.

The classical picture of the central pulse described above changes with further wave propagation toward the peripheral arteries. This is caused by a pressure damping of the basic propagating wave and by the summation of this wave with the two-phase reflected wave (Wiggers, 1952). It must be noted that the peripheral pulse curve undergoes relatively simple change and consists, very often, of only two waves: percussive and dicrotic.

In our methodology, pulse registration is used for determining individual phases of ventricular contraction. Therefore we shall not dwell on a description of the sphygmograms which are observed in the presence of this or that cardiac illness. We also shall not describe determination methods for the velocity of propagation of the pulsation wave.

III. POLYCARDIOGRAPHIC CATEGORIES AND CRITERIA IN THE SYNTHETIC ELECTROCARDIOLOGIC COMPLEX

In the chapter on ballistocardiography it was mentioned that interval R-K on the ballistocardiogram is regarded as a period coinciding with the mechanical systole of ventricles, while period R-H serves to approximate the duration of the tensile phase, and the H-K interval gives data for the /328 ejection phase. All results obtained with the use of this method are approximate and therefore cannot be used as a basis for exact investigations. For the study of the phases of the cardiac contraction, such methods as electrokymography (Heckmann, 1959; D. D. Zaretskiy, 1960; V. N. Orlov, 1960) and dynamocardiography (Ye. B. Babskiy, 1957) are also used. These methods are, however, far too technically complicated and require a relatively complex apparatus.

The method of heart probing and measuring the intracardiac pressure furnish exact information with regard to the phase structure of cardiac compression. This method however, cannot be used in wide, everyday practice, especially for the purpose of physiological investigations; it can be used only in rare pathological cases. For determination of the phase structure of the cardiac compression we utilize the method of combined investigation and analyze the obtained data according to Maass' (1954) and Blumberger's (1940) method. According to this method (Fig. 119), the tensile phase is equal to the time interval between the beginning of the electrical

excitation of the ventricles (from the beginning of wave Q, or when it is absent, from wave R of the electrocardiogram) and the start of the sphygmogram (ej), subtracting the time of passage of the pulse wave. The latter is determined by the time interval from the maximum oscillation of the II sound on the phonocardiogram to the incision of the sphygmogram. The ejection phase is determined by the time interval from the beginning of ascent of the sphygmogram (ej) to its incision. The duration of the mechanical systole is determined by the interval from the beginning of the maximum oscillations of the I sound to the beginning of the maximum oscillations of the II sound.

It is known that the tensile phase consists of two periods. The first period is that of transformation during which the intraventricular pressure is not increased and the electrical phenomena are transformed into mechanical ones. The second period is that of increased pressure during which the ventricular pressure is increased and reaches a level higher than the pressure within the main vessel (Holldack 1951). V. L. Karpman and V. S. Savel'yev (1960) use different terminology for determining these periods. They distinguish the synchronous phase of isometric contraction.

The period of transformation is determined by interval Q-I with the use of the polycardiographic method. The period of increased pressure corresponds to the difference between the phase of tension and the period of transformation.

Thus phases of heart contraction can be determined by polycardiographic investigation with the aid of the following formulas:

tensile phase = (Q-ej) - (II-incision)
period of transformation = Q-I
period of pressure increase = (tensile phase) - (Q-I)
ejection phase = ej - incision
mechanical systole = I - II
electrical systole = Q-T
heart beat rhythm = R-R

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In physiological experiments, the duration of the mechanical systole, which is to be determined, is equal to the sum of its two phases: those of tension and ejection. This, however, is not confirmed by clinical determination of these factors by means of the combined method, as the mechanical systole is somewhat shorter than the sum of its two phases. This is explained by the fact that with this method of determination, the beginning of the mechanical systole is coupled with the appearance of the I sound, while the period of transformation occurs before the appearance of this sound. In this manner a tonal equivalent of the mechanical systole is actually determined, which, according to L. A. Leshchinskiy (1960) corresponds exactly to the duration of the mechanical systole. This in turn, is found by the method of dynamocardiography. This fact causes considerable difficulties in the determination of certain functional factors (see later). It is possible to determine the electromechanical systole, which differs from the tonal equivalent of the mechanical systole, by the fact that it also includes the period of transformation and can be expressed by the formula:

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$$\text{Electromechanical systole} = (\text{I-II}) + (\text{Q-I}).$$

There is a principle difference between the mechanical and the electromechanical systoles: the mechanical systole corresponds to that period of heart contraction during which the intraventricular pressure is increased, while the electromechanical systole includes this period plus the very initial period of heart contraction. This is when individual muscular fibers begin their contraction while the ventricular pressure is not yet raised.

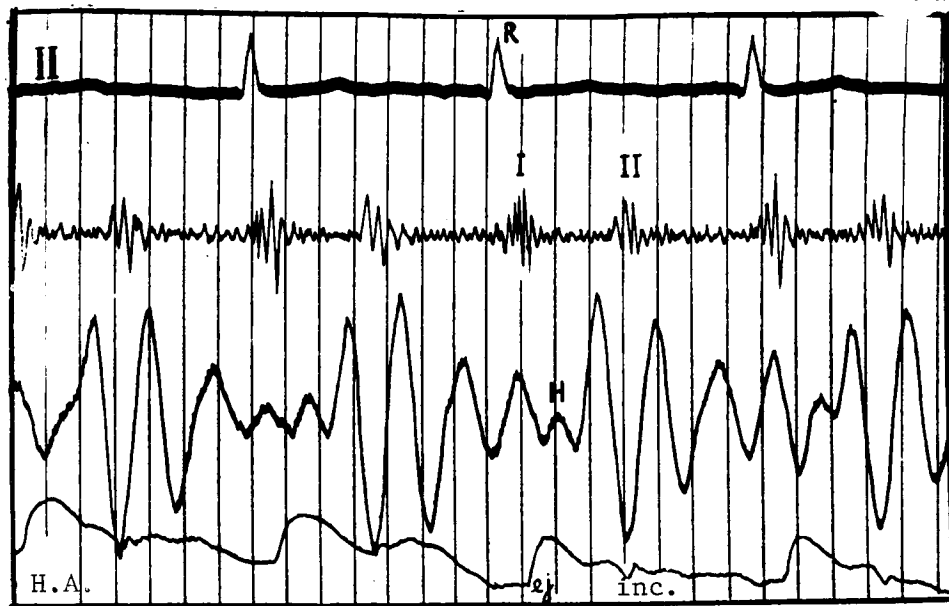


Fig. 119. Determination of phases of heart contraction according to the Maass-Blumberger method. Polycardiogram of a practically healthy individual A. N., age 16. Starting from top and going down: electrocardiogram, phonocardiogram, ballistocardiogram, sphygmogram of the carotid. Tensile phase = (Q-ej) - (II-inc.); transformation period = Q-I; period of increase of pressure = (tensile phase) - (Q-I); ejection phase = ej - inc.; mechanical systole = I-II; electrical systole = Q-T.

The information gathered from different sources with regard to the normal component values of ventricular systole is not always consistent. This can be explained by the fact that under physiological conditions considerable variability appears in the duration of these phases depending on such hemodynamic factors as pulse frequency, percussion volume, peripheral resistance, etc. The normal values which we determined after examination of a group of healthy people are given on page 365 (original text) of this book. It is interesting to note that the duration of the mechanical and ejection phases is relatively longer in women than it is in men when heart beat rhythms are equal (N. I. Yachmenev, 1959).

For the purpose of comparison and for more detailed consideration regarding duration of the individual phase of the heart beat cycle, we have compiled, in Table 10, the data by Wiggers (1945) and Willis et al. (1950).

Table 10

Phase Duration of Heart Contraction

Cardiac Phases	Wiggers	Willis et al.
Isometric contraction	0.05 sec.	0.03-0.08 sec.
Rapid ejection	0.09 sec.	0.06-0.21 sec.
Slow ejection	0.13 sec.	0.04-0.21 sec.
Total phase of ejection	-	0.17-0.32 sec.
Total systole	0.27 sec.	-
Protodiastole	0.04 sec.	{0.05-0.23 sec.
Isometric relaxation	0.08 sec.	0.08-0.30 sec.
Rapid influx	0.11 sec.	{0.04-0.74 sec.
Diastasis	0.19 sec.	-
Auricular systole	0.11 sec.	-
Total diastole	0.53 sec.	0.55-1.54 sec.
Total cardiac cycle	-	

In connection with heart diseases, changes in the phase structure of contraction depend on two factors: (1) on the character of the anatomical heart damage and, correspondingly, on hemodynamic conditions; (2) on the functional condition of the myocardium. Thus, with mitral stenosis, the tensile phase is lengthened and the ejection phase is shortened, while with aortic stenosis, the tensile phase remains almost unchanged and the ejection phase is lengthened (Figs. 120, 121).

According to S. B. Fel'dman (1960, 1961), with mitral valve insufficiency the tensile phase is either unchanged or slightly increased and the ejection phase is lengthened. With insufficiency of the aortic valve, the tensile phase is either unchanged or shortened because of the shortening of the period of increased pressure; it can also disappear completely. The ejection phase is lengthened. With hypertensive disease (Fig. 122), a very definite connection is observed between the phase-heart structure and the hemodynamic factors (Table 11). As the illness progresses, there is a definite tendency for the tensile phase to lengthen. During the first stage of the disease, the length of the tensile phase varies within normal limits, but during the second and, especially, the third stages this phase is abnormally lengthened. The increase of the tensile phase occurs because of the lengthened period of transformation. Although the period of increased pressure increases with the severity of illness, it does not exceed normal limits, and may even decrease somewhat, especially during the first and second stages. The ejection phase either remains within normal limits or is slightly lengthened. The length of this phase is almost constant during all stages. It is interesting to note that there is a definite connection between the lengthening of the tensile phase and the level of the diastolic arterial pressure.

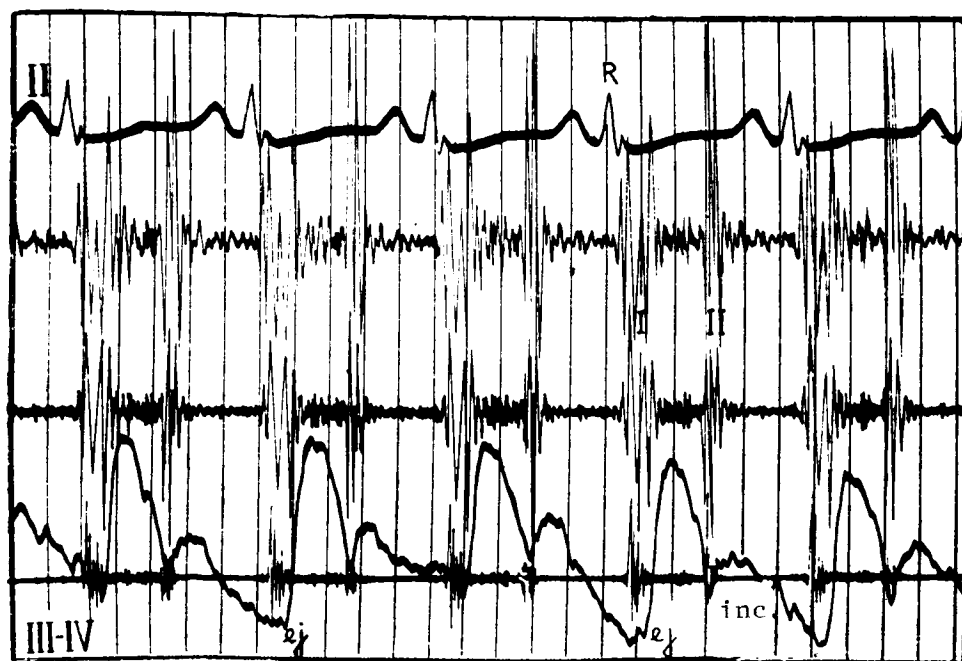


Fig. 120. Mitral stenosis. Polycardiogram of patient B. O., female, age 31. Diagnosis: mitral defect with predominance of stenosis. Tensile phase = 0.08 sec; period of transformation = 0.07 sec; period of increased pressure = 0.01 sec; ejection phase = 0.18 sec; mechanical systole = 0.23 sec; electrical systole = 0.30 sec; heart rhythm = 115 beats per min.

This is discussed by V. L. Karpman (1960, 1961) and L. A. Leshchinskiy (1960). After successful treatment of hypertensive disease, the diastolic pressure is decreased; here also a relative decrease occurs in the duration of the tensile phase. One must partially agree, with Sambhi (1960) regarding the fact that a relative or absolute lengthening of the tensile phase is one of the basic mechanisms of cardiac activity under the condition of increased load, and that the gradient of increase of the intraventricular pressure is probably the principal factor upon which the duration of the tensile phase depends.

Of considerable importance is the comparison of the actual with the theoretically obtained data for any given heart beat rhythm. As long ago /334 as 1918, Bazett submitted a formula for determining the normal duration of electrical systole as a function of the cardiac cycle (see page 61, original text). By analogy to this formula Ye. B. Babskiy and V. L. Karpman (1956) submitted the following formula for determining the normal duration of the mechanical systole:

$$\text{Mechanical systole} = 0.324 \sqrt{c}$$

where c is the time consumed by one full cardiac cycle in seconds. An observation of the dynamics of the mechanical systole and comparison of its results with theoretical values furnish additional information regarding

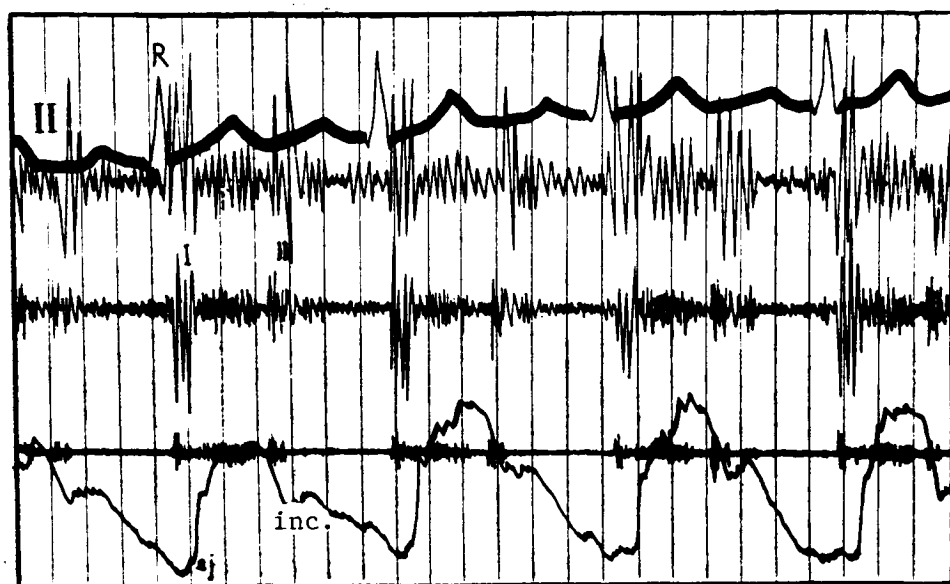


Fig. 121. Aortic stenosis. Polycardiogram of patient P. Z. female, age 34. Diagnosis: mitral-aortic stenosis. Tensile phase = 0.08 sec; transformation period = 0.07 sec; period of increased pressure = 0.01 sec; ejection phase = 0.30 sec; mechanical systole = 0.30 sec; electrical systole = 0.32 sec; heart beat rhythm = 99 beats per min.

Table 11

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Indicators of Cardiac Contraction According to Stages of Illness

<u>Indicator</u>	<u>Stages of Illness</u>		
	<u>First Stage</u>	<u>Second Stage</u>	<u>Third Stage</u>
Tensile phase (sec)	0.073 \pm 0.017	0.090 \pm 0.017	0.097 \pm 0.024
Period of transformation (sec)	0.048 \pm 0.012	0.064 \pm 0.223	0.069 \pm 0.010
Period of increased pressure (sec)	0.020 \pm 0.014	0.025 \pm 0.017	0.031 \pm 0.014
Ejection phase (sec)	0.270 \pm 0.001	0.290 \pm 0.028	0.283 \pm 0.026
Electrical systole (sec)	0.335 \pm 0.015	0.372 \pm 0.035	0.378 \pm 0.020
Mechanical systole (sec)	0.300 \pm 0.021	0.320 \pm 0.026	0.324 \pm 0.024
Internal coefficient of tension (ICT)	0.25 \pm 0.055	0.32 \pm 0.09	0.33 \pm 0.089
Corrected tension indicator (CTI)	24.3 \pm 5.95	28 \pm 6.81	29.8 \pm 6.13
Corrected ejection indicator (CEI)	91.3 \pm 5.89	91.8 \pm 5.42	87.5 \pm 6.89
Mechanical-Electrical indicator (MEI)	88.4 \pm 6.34	85 \pm 7.31	80 \pm 9.69

the functional condition of the myocardium (V. N. Brikker, 1960). Thus with mitral stenosis and insufficient blood flow into the left ventricle, the mechanical systole becomes shorter, then, after an effective commissurotomy, it is lengthened. With hypertensive disease systole is also

lengthened; electric systole lengthening somewhat more than mechanical systole. Wiggers (1952) writes that a constant lengthening of the systole by more than 0.02 sec. indicates an increase of the diastolic volume which in turn can be caused by an increase of the venous blood flow into the heart or by an accumulation of blood within the heart. This may occur in the presence of compensative pathologic conditions.

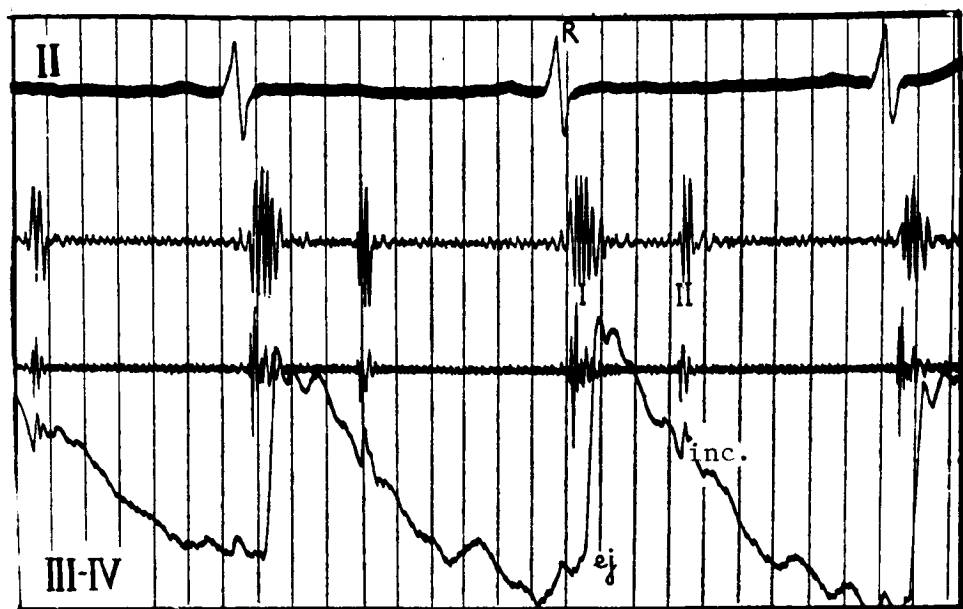


Fig. 122. Hypertensive disease. Polycardiogram of patient A. A., male, age 61. Diagnosis: hypertensive disease, stage III; arteriosclerotic cardiovascular disease. Tensile phase = 0.10 sec; transformation period = 0.08 sec; period of increased pressure = 0.02 sec; ejection phase = 0.30 sec; mechanical systole = 0.31 sec; electrical systole = 0.39 sec; heart rhythm = 66 beats per min.

Certain formulae were suggested for determining theoretical values for the individual phases of heart contraction. However, these formulae are rather too complicated and cannot be widely used. We find that comparing the duration of the individual phases with one another, it is possible to obtain very important information regarding the phase-structure of the cardiac mechanism.

With a combined investigation, it is possible to determine the following indicators of cardiac contraction:

Internal coefficient of ventricular systole (ICS) = $\frac{\text{tensile phase}}{\text{ejection phase}}$

Internal coefficient of the tensile phase of ventricular systole (ICT) = $\frac{\text{period of transformation}}{\text{period of increased pressure}}$

Corrected indicator of the tensile phase of ventricular systole (CIT) = $\frac{\text{tensile phase}}{\text{electromechanical phase}}$ in %

Corrected indicator of the ejection phase of ventricular systole (CIE) = $\frac{\text{ejection phase}}{\text{mechanical systole}}$ in %

Mechanical-Electrical indicator of ventricular systole (MEI) = $\frac{\text{mechanical systole}}{\text{electrical systole}}$ in %

The internal coefficient of ventricular systole (ICS) furnishes very important information regarding cardiac contractions in the presence of various pathological conditions and in particular, in those patients with differing degrees of myocardial insufficiency. This coefficient is, in fact, a leading functional indicator in determining the phase pattern of cardiac contraction. The importance of this indicator is considerably enhanced by the results of another indicator, namely the internal coefficient of the tensile phase of ventricular systole (ICT). This indicator provides information regarding the relationship existing between two components of the tensile phase - the period of transformation and that of increased pressure. In the pathophysiologic study of cardiac contraction great significance is assumed by this question: at the termination of which period does the alteration of the tensile phase occur; is it due to the altered period of transformation or that of increased pressure? Corrected indicators furnish general information regarding the duration of the individual cardiac phases in the total period of the mechanical systole. It must be observed that the sum of these two indicators cannot be equal to unity, because for determination of CIT we use not the mechanical, but the electromechanical systole. Mechanical systole consists only of one part of the tensile phase while the period of electromechanical systole consists of the total tensile phase. The corrected indicator of the ejection phase furnished important information on the question of the time interval consumed by the ejection phase within the total period of the increased intraventricular pressure. /335

It is a known fact that normal electrical systole begins 0.02-0.03 seconds before the mechanical systole and that its duration is longer by 0.04 sec. In pathological cases, this relationship may be considerably altered. Thus, for example, with mitral stenosis the mechanical systole is considerably shorter than the electrical systole. (V. S. Moiseyev 1960), and with hypertonia, the cardiac systole is generally lengthened; but in this case, the electrical systole is considerably more lengthened than the mechanical systole (Table 11). In 1956, Kunos and Garan suggested a way to determine the degree of contraction of the left venous aperture with the use of values of the electromechanical coefficient (MEI). However, they took interval I-os and not that of I-II for the duration value of the mechanical systole, i.e. they also included the starting period of the diastolic phase which corresponds to the period of the isometric ventricular relaxation. The normal value of the true (from the physiological standpoint) electromechanical indicator is easily determined by Bazett's (1918) formula and by those of Ye. B. Babskiy and V. L. Karpman (1956), i.e.,

$$MEI = \frac{0.324 \sqrt{c}}{0.37 \sqrt{c}}$$

(Normal values of all indicators are given on page 365).

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It must be taken into consideration that shifting of these indicators to either side can occur as a result of alterations in either the numerator or denominator. Therefore it could happen that with entirely different alterations in cardiac phases, one may obtain the same values for the indicators.

In order to avoid such difficulties one must make a careful analysis of the absolute values of the individual components of cardiac contraction.

We think that the terminology which we have suggested corresponds to the context of these indicators. It must be noted that in the literature, the corrected indicator of ejection (CIE) is sometimes known as the intersystolic indicator (V. L. Karpman). We cannot use this designation because the actual corrected indicator of tension is also an intrasystolic indicator.

Moreover, in ballistocardiography, the coefficient $\frac{\text{interval H-K}}{\text{interval R-K}}$ in % is also

designated as an intrasystolic coefficient, although, as a rule, there is no coincidence between the value of this coefficient and that of the ejection indicator, because the ballistocardiographic intervals H-K and R-K do not correspond exactly to the phase of ejection and to the mechanical systole.

IV. CONCLUSION

The method of combined investigation, with the use of electrocardiology, phonocardiology and arterial sphygmography, is technically simple and easily available to any electrocardiologist. This method permits the possibility of studying the mechanical cardiac systole and its phase structure, and aids in the determination of a series of indicators which have great significance in clarifying the functional condition of the heart. The use of this method considerably enriches the arsenal of electrocardiology because it permits deeper insight into minute mechanisms of cardiac contraction and, indirectly, aids in obtaining an understanding of the condition of intracardiac hemodynamics. Polycardiographic investigation widens considerably the possibilities of synthetic electrocardiology and, in conjunction with other methods, contributes to obtaining a complete and clear picture of the cardiac mechanism in the presence of various physiological and pathological conditions.

PART FOUR

PRACTICAL PROBLEMS IN SYNTHETIC ELECTROCARDIOLOGY

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I. INTRODUCTION

In the preceding chapters we have given a detailed description of the methods applied in synthetic electrocardiology and have shown the role and importance of each of these methods for various aspects of cardiac activity. /339

For better clarity and facility of exposition, the electrical and mechanical phenomena were analyzed separately, although such division could only be provisional. The final purpose of synthetic electrocardiology, however, consists of an endeavor to collect the results obtained by all methods and to synthetically form a general functional presentation and also a partially anatomical presentation of the condition of the heart.

This task can be performed using the following schematic plan as a point of departure. This plan contains the evaluation and significance of each of the methods applied in the determination of one or another condition.

II. STUDIES OF CARDIAC FUNCTION

1. Automatism

Automatism is studied by the method of electrocardiology; for the purpose of this study no other methods are of any significance whatsoever. It is true that by applying these methods, it is possible to determine the number of heart contractions per minute and to establish the heart rhythm. It is also possible to discover the presence of sinusoidal tachycardia, bradycardia or arrhythmia. In the presence of tachycardia, one may find shortening of the systole on the phonocardiogram and also summary gallop, and in the presence of bradycardia one may discern the III sound. Despite this, these methods are unable to add any significant data other than those obtained by electrocardiology, the more so in the presence of the heterotonal rhythms especially under the condition of nodal rhythm or that of the phenomenon of the migrating pacemaker of heart beat rhythm.

2. Conduction

In the study of conduction, the method of electrocardiology assumes a leading significance both in normal and pathological conditions. In determination of the sinusoidal auricular and intra-auricular blocks, as well as in that of the syndrome of premature excitations of ventricles, electrocardiology alone is capable of providing exhaustive data. With other types of disrupted conduction, different techniques may serve for acquiring certain additional, indirect data, the recognition of which is of but little value. For example, in the case of a pronounced partial atrioventricular block of the first type, it is possible to see, on the phonocardiogram, the auricular sound and the weakening of the I sound. For the second /340

type, it is possible to discern a gradual weakening of the I sound and its withdrawal from the auricular sound during a series of cycles of cardiac activity until that particular cycle where sounds I and II are absent (Wenckebach-Samoylov period). With total atrioventricular block, the phonocardiogram records a variability in the intensity and shape of the I sound, whereupon, as the auricular and the ventricular contractions coincide and on the electrocardiogram, wave P runs together with the QRS complex, the phonocardiogram shows an intensified I sound (Strazhesko cannon tone); if, however, wave P and QRS complex are very close together on the electrocardiogram, the phonocardiogram displays the pattern of a split I sound. With this type of block, one is able to discern the auricular sounds which correspond to the frequency of the auricular contraction. The I and II heart sounds originate in accordance with the rhythm of the ventricular contractions.

The method of vectorcardiology provides richer data in the case of bundle branch blocks. These data enrich quite effectively the possibilities of synthetic methodology. As we have already mentioned, the significance of this method is increased especially in the combined case of bundle branch blocks with a myocardial infarction because in this case, an electrocardiographic analysis alone may prove to be very insufficient for the purpose of correct diagnosis. In the presence of such blocks, the splitting of the I and the II sounds may appear on the phonocardiogram due to asynchronism between the beginning of contraction in the two halves of the heart.

3. Excitability

Excitability can also be studied by electrocardiography. The importance of this method is undisputable in the topical determination of the genesis of extrasystolic impulses. The ballistocardiogram and phonocardiogram reflect the hemodynamic pattern of the extrasystolic contraction. In this case the degree of intensity of the corresponding waves and sounds basically depend on one common mechanical factor - on the degree of the prematureness of the extrasystolic impulse. On the ballistocardiogram, the extrasystolic complex is altered and deformed with diminution of the wave amplitude due to insufficient blood flow into the ventricles. The complex which follows the compensatory pause is well differentiated and, due to the lengthy and ample blood flow at that time, the waves are of greater amplitudes than the waves of other complexes.

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The phonocardiogram shows a shortened I-II interval for an extrasystolic contraction. The I sound is intensified as a result of a longer stroke and harder closing of the atrioventricular valves when blood flow is slow into the ventricles. The amplitude of the II sound is decreased because at that time the pressure is decreased; during the ejection phase there is only a small flow of blood into the vessels. In this case, if the prematureness is considerable, the II sound may even disappear altogether. The second sound of the post-extrasystolic contraction is intensified as a result of the ample blood flow into the ventricles during the lengthy compensatory phase. With ventricular extrasystoles, the splitting of sound I and more often that of II, occurs because of the premature closing of valves in that half

of the heart where the premature impulse occurs.

With a paroxysmal tachycardia of supraventricular or ventricular origin, ballistocardiography does not furnish any information. For a condition of such frequent heart contraction this method has no validity. The same may be said regarding phonocardiography - with such a frequent rhythm the individual cardiac sounds overlap each other.

Electrocardiography is of the utmost importance in determining tremor and fibrillation of the auricles. In vectorcardiographic investigation, it is possible to observe certain waviness of the isoelectric line; in one of our own cases this waviness was of considerably high amplitude. The hemodynamic part of this arrhythmia is well pronounced on the phonocardiogram; an extensive variability of heart sounds is observed, whereupon with a premature cardiac contraction, the I sound is intensified and the II sound becomes weaker. With a retarded heart contraction, the opposite phenomena are observed. It must be emphasized that there exists an extensive variability in noise phenomena - especially of presystolic noises. In the absence of the tachysystolic type of auricular fibrillation, such variability of complexes is also observable on the ballistocardiogram; but in the presence of such fibrillation, ballistocardiography is almost ineffective. The data provided by a ballistocardiogram do not always indicate a large impairment of the myocardial contractive function in the presence of auricular fibrillation.

4. Contractility

Contractility is studied by means of ballistocardiography, phonocardiography and combined polycardiographic investigation. In this connection, no significant importance should be ascribed to the methods of investigation of electric cardiac activity; we also consider as doubtful the significance of the systolic indicator and the determination of the absolute electric heart systole as well as its comparison with the values of Bazett's formula. The reason for this is that the duration of the electric systole depends on the propagation of the excitation process which in turn depends not so much on the anatomical condition of the contractive part of the myocardium as it depends on the condition of the conduction system, myocardial excitability, the dynamic equilibrium of the neuro-humoral heart regulating apparatus and on a series of other factors which have no direct or immediate connection with the contractile energy of the myocardium. /342

Ballistocardiography gives general aspects regarding the condition of the contractile heart function and its dynamics, in a particular direction. This method introduces an important share of functional direction into the complex of synthetic electrocardiology. It must be noted that by use of this method, it is possible to recognize very early myocardial insufficiency even when it is not yet possible to clinically detect any definite symptoms of heart insufficiency.

We want to underline the following results obtained with the use of this method.

a. With definite, but not very pronounced alterations of the systolic waves during the exhalation phase, when the general pattern of the curve is not yet changed, and the wave amplitude is not yet diminished, it is feasible to suspect a latent progression of heart insufficiency.

b. An increase and deformation of wave H, and the appearance of early M-like complexes must be regarded as a symptom of decreased ability of the heart to function, even in the presence of a good general curve pattern and the absence of clinical manifestations of heart insufficiency.

c. It is possible to connect an increase of interval R-H with a weakening of the heart's ability to contract whereupon, if this increase occurs in conjunction with pathological alterations in the systolic complex of the curve, it assumes a still greater significance in the diagnosis.

d. Determination of the degree of pathological discrepancies of the ballistocardiogram with Brown's criteria, with Lin'-Chen's supplement, assumes certain significance. The first degree of these discrepancies may fail to indicate the presence of the cardiac insufficiency, but the second, and the more so the third or fourth degrees of discrepancy show that it is possible to doubt the weakness of the myocardium; and that there is a direct, although not necessarily constant, connection between the degree of pathological discrepancy and the degree of cardiac insufficiency. In certain cases it may happen that there is no observable relationship between the results found by ballistocardiography and the functional conditions of the heart. This may be explained principally by the fact that (as we have mentioned before) in a ballistocardiogram, the curve reflects not only heart contraction, but also the mechanism of the entire vascular system, whose many functional indicators exert considerable influence on the character of the curve. It is known that there may be a lack of correspondence between the functional condition of the heart and the condition of the vascular system. In this connection also one must not forget the condition of the respiratory system, because in the classification of pathological deviations of the ballistocardiogram a large portion is ascribed to respiratory oscillations of the systolic waves.

In all these cases, methods of electrovectorcardiography and phonocardiography are capable of giving only those data which are characteristic of an individual infirmity.

Although it is possible, in a certain sense, to determine ballistocardiographically the length of the mechanical systole and the duration of the tensile phase and the ejection phase, the phase structure of cardiac contraction is studied considerably better by the use of polycardiographic investigation. In addition, the latter is of great significance in determining a series of important functional indicators (ICS, ICT, CIT, CIE, MEI). So far we cannot yet speak of a direct and clear connection between the data of these indicators and the condition of the myocardial contractile function. This is so because the changes in value of these indicators principally depend on those internal and external cardiac hemodynamic deflections which characterize the peculiarities of blood circulation char-

acteristic for a given heart disease; for example, with mitral stenosis and insufficiency of the aortic valve, these indicators assume entirely different values independent of the myocardial condition. Our next objective will consist of an endeavor to discover those changes in these indicators which can serve as criteria for diagnosis of cardiac insufficiencies in conditions of hemodynamic peculiarities pertaining to various heart diseases.

III. DETERMINATION OF ISOLATED HEART CONDITIONS

1. Heart position

Heart position is determined by the method of electrocardiology. This determination is based on Goldberger's (1954) criteria according to which the heart rotates about three principal axes. In practice it is sufficient to consider the rotation about a single front-rear axis, using for this purpose the position of the heart's electrical axis and Wilson's criteria regarding electrical heart positions. Vectorcardiography is disregarded, although this method may also serve to obtain certain data. Other methods of synthetic electrocardiography are of no value in the determination of heart positions.

2. Ventricular and Auricular Hypertrophy

Ventricular and auricular hypertrophy is studied, basically, by the method of electrocardiography. In the first place the changes due to hypertrophy are studied and also the dynamics of the electrical heart equilibrium; the methods of study of electric cardiac activity are most suitable in this connection. In our own practice we came to the firm conclusion that electrocardiography furnishes a very rich arsenal of indicators for diagnosis of isolated or combined ventricular hypertrophy and it may be said without exaggeration that among all the methods of study of heart hypertrophy, first place must be allotted to this method. Electrocardiography is especially valuable for a dynamic study of the progress regression of hypertrophy as a result of overloading or unloading of the affected heart section. /344

The method of vectorcardiography (or vectorelectrocardiography) supplements the findings of electrocardiology in the discovery of ventricular hypertrophy. The significance of this method consists basically of the following:

a. Vectorcardiography presents an excellent study of the progressive states of ventricular hypertrophy. Thus, with the development of hypertrophy, the QRS loop gradually orients itself toward the side of the hypertrophic ventricle; with hypertrophy of the left ventricle this orientation moves to the left, to the rear and upward, while with hypertrophy of the right ventricle - to the right, forward and upward. In addition to this, there is a gradual increase of the angle of divergence between the maximum vectors of loops QRS and T; in more pronounced cases of hypertrophy loop T assumes a discordant position. Together with these phenomena, there appears an in-

crease in the area of loop QRS and a reduction of its closeness.

b. More valuable data are provided by vectorcardiography in the initial stages of ventricular hypertrophy, especially of the right ventricle. This question is not yet sufficiently explored, but the validity of vectorcardiography is quite definite, because in the late stages of hypertrophy, electrocardiography furnishes more than sufficient criteria, while vectorcardiographic data reflect only those dystrophic and sclerotic changes which develop in the hypertrophic myocardium.

c. On the basis of our own electrovectorcardiographic data, we assume that the method of vectorial analysis of electric forces within the heart may have an important quantitative value in the study of the reverse development of hypertrophy after elimination or decrease of the cause of the hyperfunction of the heart section under consideration.

We do not ascribe any considerable significance to other methods of synthetic electrocardiology so far as the study of hypertrophy of various heart diseases is concerned; the data obtained by these methods have no diagnostic value for hypertrophy proper since they pertain to basic and associated diseases that cause the development of the hypertrophy.

Ventricular hypertension is studied by the electrocardiographic method (this electrocardiographic category we separate provisionally from the category of hypertrophy, considering the difference of their manifestations on the electrocardiogram). In this case also, vectorial analysis may furnish supplementary data: an increase of divergence of the angle between QRS and T, and the appearance of vector RS-T.

3. Coronary insufficiency

In the study of coronary insufficiency, the synthesis of data obtained with the use of electro-, vector- and ballistocardiography acquires a very significant importance. Phonocardiography is unable to furnish any significant data this condition excepting the fact that a myocardial infraction is accompanied by the appearance of the III sound which, prognostically, is a bad symptom (L. M. Fitileva, 1962). We want to underline the following advantages of the synthetic method. /345

a. In the very early stages of coronary insufficiency, the electrocardiogram and the vectorcardiogram may be devoid of any alterations. During this period, when the electrical heart activity is not yet noticeably altered, there is already an impairment of the heart's contractile function, and ballistocardiographic investigation discovers alterations which, though not pronounced, indicate pathological conditions of the myocardium.

b. In the early stages of coronary insufficiency, the electrocardiogram displays a concordant upward deflection of segment RS-T and an increase in wave T. The shape of these components has a significant diagnostic importance. Segment RS-T becomes shortened and often acquires an arc-like shape, while wave T loses its normal asymmetry and its steeply ascending and de-

scending sides form a relatively sharp apex. In addition, electrovector cardiographic data may indicate an alteration of the QRS-T angle and the appearance of the RS-T vector. The ballistocardiographic investigations reveal an impairment of the myocardial contractile function.

c. Major importance must be ascribed to the electrocardiographic method in the determination of localization of myocardial infarction, and also in the dynamics of their development and propagation. We think, however, that the synthesis of data obtained by a series of methods, tends to widen the physician's range of vision when he examines patients suffering from a myocardial infarction. The appearance of the infarct vector aids considerably in the refinement of infarct localization. In addition, as was already indicated, vectorcardiographic investigations contribute considerably to the recognition of rear-diaphragmal and rear-lateral infarction and of the infarction appearing in conjunction with bundle branch blocks. In the presence of myocardial infarction, ballistocardiographic investigation increases the cardiologist's scope of information. It furnishes a point of departure towards the solution of the prognostically important question regarding either restoration or further impairment of the condition of the myocardium and that of the whole cardiovascular system.

4. Electrocardiographic Method

The electrocardiographic method also serves to determine nonlocalized, scattered changes in the auricular or ventricular myocardium. We did not make a separate presentation concerning these alterations, although data on this subject are found in almost every section of this book. In the presence of such changes, the existence of impairments to myocardial contraction is made clear by the method of ballistocardiography.

5. Normal Cardiac Sounds

There is a change in the acoustical pattern of normal cardiac sounds /346 in the presence of any kind of pathologic heart condition. The sound phenomena appearing as a result of cardiac contraction are connected not so much with the motion of heart muscles as with hemodynamic displacements and valve motions. For these reasons, in our own methodology, phonocardiography is regarded not only as an important method for the study of cardiac sound manifestations, but also as a method by which it becomes possible to visualize the displacements within intracardiac hemodynamics and, in a certain sense, to pass judgement regarding the functional condition of the myocardium. In our work we became convinced that a good phonocardiographic analysis together with results obtained by other methods can furnish rich information. This is especially true under the following conditions.

a. In the presence of acquired or congenital heart defects, phonocardiography is of great importance for refined determination of the anatomical heart condition. Electrocardiography provides data regarding hypertrophic indications of various heart sections and determines the degree of impairment to the myocardial blood supply of the various affected sections. Ballistocardiography provides data on the myocardial contractile function. By synthesiz-

ing data provided by these three methods it becomes possible to make a correct decision with regard to any indicated surgical treatment.

b. In the presence of mitral stenosis phonocardiography and electrocardiography tend toward refined answers to two important problems: the degree of the existent stenosis and simultaneously, the presence or absence of regurgitation.

Phonocardiography furnishes data regarding: intensity of the I sound on the heart apex; intensity and splitting of the II sound on the pulmonary artery; intervals Q-I and II-os; optimum region for auscultation of the diastolic noise; intensity and shape of the presystolic noise; presence of the systolic noise together with its changes, with patient recumbent on left side; and change of the systolic noise at the zero point. Electrocardiographic data comprises such factors as: degree of overloading on the right ventricle and the left auricle; shape of the wave P, especially in leads I, II, aVr, aVF and V in the absence of auricular fibrillation; and types of ventricular complex, especially in leads VR and V_{1,2,6}. On the basis of this information we distinguish three degrees of stenosis: moderate, medium and considerable. In most cases, the results of our investigations were verified on the operating table. It is interesting to note that the degree of ballistocardiographic deviation does not always reflect the degree of stenosis and may be observed in various combinations.

In determining the presence of regurgitation combined with stenosis, we take into consideration the three following occurrences: (1) indication of left ventricular hypertrophy on the electrocardiogram, when there is already a pronounced pattern of right ventricular hypertrophy; (2) the unamplified I sound coupled with the regurgitating systolic sound which does not change its intensity at the zero-point; (3) presence of the III sound on the phonocardiogram.

c. Synthesis of electrocardiologic data does not easily provide the necessary accuracy for determining the degree of aortic stenosis. When the stenosis is isolated, this is possible to a certain degree by examining the patterns of hypertrophy and overload of the left ventricle. The shape and localization of the apex of the systolic noise of ejection may also assist in this determination. However, in our observations, in most cases the aortic stenosis was combined with a mitral defect. This considerably distorts the hemodynamic pattern of the aortic defect and correspondingly changes the electrical and mechanical cardiac activity. This problem requires further study.

d. In postoperative observations, after mitral commissurotomy, analysis using synthetic electrocardiology yields very valuable data. Electrocardiography shows gradual deloading of the right ventricle and left auricle coupled with gradual increased loading in the left ventricle. This period lasts for several months. Quite often, however, there is no parallel observed for a long time between the clinical condition of the patient and the pattern of electrical cardiac activity (see page 133). In such cases, the success of surgery is judged by a diminution of the amplitude

of the I sound on the heart apex and of the II sound on the pulmonary artery; also by a decrease of interval Q-I, by the disappearance (or diminution) of the diastolic noise, (especially the amplified presystolic noise) and by the absence of the systolic noise, (although there is sometimes a lack of direct connection between the changes in the acoustical pattern and the clinical condition of the patient.) Moreover, ballistocardiography may also indicate an improvement of the myocardial contractile function.

6. Pathological Conditions

In the presence of various pathological conditions, synthetic electrocardiographic methodology assists in the solution of problems of a clinical nature and fosters new ideas for research. For illustration we shall give examples of our own experiences in cases of hypertensive disease. Our detailed observations of a large group of patients showed characteristic changes in their electrocardiograms, vectorcardiograms, ballistocardiograms and phonocardiograms. Such changes were also observed in the combined investigations. These results were discussed in the respective chapters of this book, and we do not wish to repeat ourselves. However, simultaneous analysis of data obtained by different methods resulted in a series of interesting clinical observations, of which the following are worthy of special consideration.

a. By analyzing the ballistocardiographic data, it was found that quite often there is no relation between the clinical stage of a disease and the degree of pathologic deviations on the curve. Thus, sometimes the third or even the fourth stage of the pathologic deviations (according to Brown) in the first stage of the disease. We experienced difficulty in explaining this fact, but the analysis of the electrocardiographic data indicated that, in general, coronary insufficiency increases with growing severity of the disease, but that there is no full parallel between the degree of coronary insufficiency and the stage of the disease. This seeming contradiction was explained. A relationship was found between the condition of the contractile function and the nutritive blood supply of the myocardium. /348

b. In our observations we did not always find a parallel between electrocardiographic and ballistocardiographic data. In the corresponding chapter we have tried to explain this fact (see page 273, orig. text). It is possible to assume, however, that with hypertensive disease, changes in the ballistocardiac forces are connected to other factors. This urged us to study the indicators of respiratory function. It was found indeed, that patients with this ailment had an impairment of the respiratory function which may also be of importance in explaining the described controversy.

c. The phonocardiogram of patients suffering from hypertensive disease quite often displays an amplification of the II sound and non-intensive systolic noise on the pulmonary artery. This fact called for study of the question of whether hypertensive disease also causes an increase of pulmonary

hypertension. For this reason we applied the whole complex of synthetic electrocardiology and determined a series of indicators regarding external respiration and the velocity of blood flow in the pulmonary circulation. We used neither the direct method for evaluating pressure within the right heart nor pulmonary artery catheterization of the heart, since, at that time, it was not technically possible, but also because it is preferable not to apply such a relatively complex and unsafe method of examination of patients where there is no drastic need for it.

Phonocardiographic data indicates the presence of pulmonary hypertension; this was confirmed by the examination results of external respiration and the velocity of blood flow in the pulmonary circulation. This, however, was contradicted by the fact that electrocardiographic and vectorcardiographic examinations did not indicate the presence of hypertrophy of the right ventricle (ballistocardiography could not furnish any additional information). We think that the available criteria as yet do not allow the use of this method for the separate study of the functional condition of the left and the right halves of the heart. After having synthesized all obtained data, together with the pathoanatomical data obtained by other authors, we arrived at the clinical hypothesis that hypertensive disease causes the development of a certain degree of right ventricular hypertrophy. Regarding the question of why this hypertrophy is not reflected electrocardiographically, we came to the following conclusion. It is known that hypertrophy of either ventricle is reflected on the electrocardiogram because of a deviation from the /349

normal coefficient $\frac{\text{mass of the left ventricle}}{\text{mass of the right ventricle}}$. With hypertensive disease the

electrical pattern of hypertrophy of the right ventricle cannot be discerned, because due to the presence of pronounced hypertrophy of the left ventricle, the coefficient value for the mass of the left ventricle is pronounced. Even with a slight degree of hypertrophy of the left ventricle, again hypertrophy of the right ventricle cannot be discerned because there are no discernible changes in the dynamic balance between electrical forces of both ventricles. In such cases the electrocardiogram cannot reflect the hypertrophic pattern of either ventricle. We encountered such examples in practice.

d. Phonocardiograms of certain patients showed the IV sound, or the precardiac sound, which occurred after wave P and which did not blend with the I sound. With considerable hypotension this precardiac sound approached the I sound or blended with it. Synthetic electrocardiologic methodology offered the possibility of corroborating more accurately the fact that the appearance of the IV sound, during the latter period of blood flow into the ventricles, is caused by weakness or hypertension of the left ventricle. Under therapeutic treatment the condition of the myocardium was improved, and the IV sound disappeared. This conclusion coincides with opinions expressed in the literature.

7. Synthetic Electrocardiology

The use of synthetic electrocardiology permits a clinical comparison of

the electrical and mechanical manifestations of cardiac activity. With such an approach it is possible to ascertain the interdependence of the characteristic segments of various curves. Thus, on the basis of the electrical complex of the ventricular systole, one is able to define the following events: wave H appears on the ballistocardiogram 0.07-0.08 sec. after the apex of wave R on the electrocardiogram. Wave IJ is formed at the middle of the electrical systole; wave K appears toward the end of wave T; waves L, M and N are formed during interval T-P; 0.04-0.06 sec. after the start of wave Q the first maximum oscillation of the I sound appears; toward the end of wave Q, the first maximum oscillation of the II sound appears; in the early period of T-P, the III sound is observed, while during interval P-Q of the next complex the IV sound appears. 0.08-0.10 sec. after wave Q, a percussive wave of the carotid begins, while 0.02 sec. after the beginning of the maximum oscillation of the II sound, an incision forms in the sphygmogram. The significance of all these intercor- /350 relations can be visualized by remembering the following basic statements:

Start of wave Q = start of the electrical systole;
End of wave T = end of the electrical systole;
Interval (T-P) + (P-Q) = diastole;
First optimum oscillations of the I sound = snapping of atrioventricular valves;
Optimum oscillations of the II sound = closing of the semilunar valves;
III and IV sounds = sounds of blood influx;
Start of the sphygmogram ascent = start of ejection;
Sphygmogram incision = end of ejection.

Such a diagram permits complete electrocardiologic examinations under any physiological or pathological conditions (for example, in studying the effects produced on the heart by various factors, in determining the effectiveness of a new apparatus, in the presurgical preparation of patients, in the study of the surgical treatment, etc.). Thus, this diagram may be used with any kind of clinical examination. At first we thought of presenting another scheme as well for analyzing and synthesizing electrocardiological data from the point of view of individual determination of the anatomical and functional condition of the heart. Later, however, we came to the conclusion that, from a theoretical standpoint it would be difficult to separate these two aspects of cardiac condition. Because of this, such an approach could not contain an anatomical diagnosis of cardiac pain. The physician's problem consists only of describing the changes found by examination and, possibly, of making a series of electrophysiological and biophysical inferences; only after this can a clinician, or perhaps, the electrocardiologist himself, make a complete diagnosis of the patient's illness on the basis of the clinical electrocardiological parallel by considering all data obtained by supplementary laboratory and instrumental methods.

For practical use of a synthetic electrocardiological laboratory we recommend a special form. It is made up in such a way that any physician can obtain a complete picture of the condition of heart activity by placing his notations on the form.

IV. THE FORM FOR SYNTHESIZING AND ANALYZING THE DATA
FROM ELECTROCARDIOLOGIC INVESTIGATIONS

The following form has been developed by us based on the principles of
synthetic electrocardiology:

INSTITUTE OF CARDIOLOGY AND HEART SURGERY OF THE
ARMENIAN SSR ACADEMY OF SCIENCES

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LABORATORY OF SYNTHETIC CARDIOLOGY

Investigation No. _____

Date _____

Patient _____

Hospital, case history No. _____ Section _____

Ambulatory _____

Diagnosis _____

Age _____ Weight _____

Purpose of examination _____

City of Yerevan

A. ELECTRICAL HEART ACTIVITY

/352

1. Electrocardiography

a. Quantitative indicators

Duration (for lead II):

P _____ sec.

Segment P-Q _____ sec.

QRS _____ sec.

RR _____ sec. (Rhythm _____ per minute).

QRST _____ sec. (according to Bazett's norm for this rhythm _____ sec.)

Amplitude (show only for those leads where it can have a diagnostic significance):

P _____ mm

Q _____ mm

R _____ mm

S _____ mm

T _____ mm

U _____ mm

Total Voltage _____ m

Electrical axis _____

Ventricular gradient _____

Time of appearance of internal deviation:

V₁ _____ sec.

V₅ _____ sec.

V₂ _____ sec.

V₆ _____ sec.

b. Functions of automatism, excitability and conductivity:

c. Qualitative indicators

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Lead \ Component	Component				
	P	QRS	Segment RS-T	T	U
I					
II					
III					
aVR					
aVL					
aVF					
V ₁					
V ₂					
V ₃					
V ₄					
V ₅					
V ₆					

d. Functional probes:

2. Vectorcardiography

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a. Precordial method according to Akulinichev

Loop Characteristic \ Projections		I	II	III	IV	V
Shape	QRS					
	T					
Arrangement	QRS					
	T					
Magnitude of Maximum vector	QRS					
	T					
Maximum width	QRS					
	T					
Area, cm ²	QRS					
	T					
Course	QRS					
	T					
Angle diversion QRS-T						
Loop P (with special plotting)						

Spatial pattern of Vectorcardiogram:

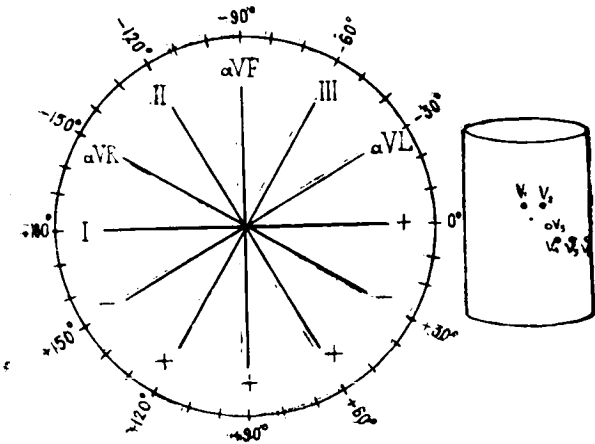
b. Cube method according to Grishman

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Frontal	Sagittal	Horizontal

Spatial pattern of vectorcardiogram:

3. Spatial Vectorcardiography



vector QRS _____
vector RS-T _____
vector T _____
angle QRS-T _____

B. MECHANICAL HEART ACTIVITY

/356

1. Ballistocardiography

(show the technique of plotting).

a. Quantitative indicator.

Intervals in seconds

R-H	R-I	R-J	R-K	R-L	H-I	I-J	J-K	K-L	H-K	I-K

Amplitude in mm				Coefficients			Respiratory Coefficient	Ballistographic Index	Intrasystolic Indicator
HI	IJ	JK	KL	$\frac{HI}{IJ}$	$\frac{JK}{IJ}$	$\frac{KL}{IJ}$			

b. Qualitative indicators (description with calm breathing, on the level of sustained inhalation and exhalation, degree of pathology according to Brown).

c. Functional probes

2. Phonocardiography

(show the technique of plotting)

With auscultation:

a. Quantitative indicators (show the area)

Duration: I sound _____ sec.

II sound _____ sec.

Interval I-II _____ sec.

Interval Q-I _____ sec.

Interval II-os _____ sec.

Amplitude: I sound _____ mm

II sound _____ mm

Frequency: I sound _____ oscil./sec.

II sound _____ oscil./sec.

b. Qualitative indicators

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Region Sound Manifestations	Mitral	Aortic	Tricuspid	Pulmonary	Supplementary
I sound					
II sound					
III sound					
IV sound					
Supplementary sounds					
Gallop rhythm					
Noise phenomena					

c. Functional probes.

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3. Combined Investigation

(electro-, phonocardiography and sphygmography of the carotid)

Duration: Interval Q-I _____ sec.
 Interval Q-ej. _____ sec.
 Interval II-inc. _____ sec.
 Interval ej-inc. _____ sec.
 Interval I-II _____ sec.
 Interval Q-T _____ sec.
 Tensile phase _____ sec.
 Period of transformation _____ sec.
 Period of increased pressure _____ sec.
 Ejection phase _____ sec.

Internal systole coefficient (ISC) _____

Internal coefficient of tensile phase (ITC) _____

Corrected indicator of tension (CIT) _____%

Corrected indicator of ejection (CIE) _____%

Mechanical-Electrical indicator (MEI) _____%

CONCLUSION

/360

Laboratory Manager

Remarks _____

V. NORMAL VALUES FOR SYNTHETIC ELECTROCARDIOLOGIC INDICATORS

The following normal indicators of synthetic electrocardiography were established from our observation of two groups of healthy individuals: one group of ages 17 to 30 (number in the upper row for each indicator), the other group of ages 30 to 55 (numbers in the lower row). All observations were made simultaneously. Heart rhythm varied within the limits of normal sinusoidal rhythm, averaging from 60 to 70 beats per minute. The heart position was found to be basically intermediate. The following tables contain the maximum and minimum values for each variational row, the arithmetic mean (M) and the mean quadratic deviation (G).

A. ELECTRICAL CARDIAC ACTIVITY

I. ELECTROCARDIOGRAPHY

Table 12

Wave Amplitudes mm.															
Lead	P			Q			R			S			T		
	Max.	Min.	M ±	Max.	Min.	M ±	Max.	Min.	M ±	Max.	Min.	M ±	Max.	Min.	M ±
I	1	0.4	0.7±0.09	0.4	0	0	12	3	6±0.8	5	0.5	2±0.53	3.5	1	2.1±0.22
II	2	0.9	1.2±0.15	1	0	0.4±0.1	17	4	10±1.3	3	0	1±0.3	6	1.4	2±0.2
III	1	0.9	1.3±0.1	0.8	0	0	11	3	8±0.8	2.5	0	1.2±0.3	4	1.9	3±0.35
aVR	1	-0.2	0.5±0.1	2	0	0.7±0.17	15	2	7±1.1	3	0	—	4.9	-1.4	2.5±0.2
aVL	1	0.5	0.8±0.09	1.5	0	0	7.5	0.5	4±0.7	4	0	1.4±0.4	2	-0.5	1.3±0.5
aVF	1.5	-0.2	-0.9±0.08	13	2	7±0.8	2	0	0.9±0.3	0	0	0	-5.2	-1	1±0.2
V ₁	0.5	-0.4	-0.8±0.06	9	2	5.5±0.5	3	0	0.8±0.2	0	0	0	-3	-1	-2.3±0.3
V ₂	0.9	0	0.3±0.07	1.3	0	0	7	0.5	3±0.6	3	0	3.1±0.8	1.8	-0.5	-2±0.14
V ₃	0.5	0	—	2.5	0	0	9	0.5	3±0.6	3	0	1±0.3	2	-0.5	0.8±0.2
V ₄	2	0.5	0.8±0.1	1.2	0	0.4±0.1	15	1	8±1.1	3	0	—	4	0.8	0.7±0.2
V ₅	2	0.6	1±0.08	0.5	0	0	10	1	5±0.7	5.5	0	1±0.3	3.5	0.8	2±0.2
V ₆	0.5	-1	-0.2±0.15	0	0	0	5.2	0.9	1.8±0.3	15	3	8±1.0	2	-3.8	-1.6±0.4
V ₇	±0.5	-1	-0.5±0.1	0	0	0	3	0.5	1.5±0.2	10	3	7±0.7	1	-2	-1.2±0.15
V ₈	1	0	0.6±0.09	0	0	0	12	0.9	6±1.3	16	3	9±1.2	8.1	-2	3.3±0.08
V ₉	0.8	0	0.3±0.07	0	0	0	10	0.9	5.5±0.7	14	3	9.5±1	11	2	4.5±0.7
V ₁₀	1	0	0.6±0.08	0	0	0	19.1	1.1	8±1.3	16	0	11±1.5	13.3	-2	4.7±1.1
V ₁₁	1	0	0.6±0.07	0	0	0	12.5	1.1	8±0.8	16	3	11±0.9	10	2.2	6±0.6
V ₁₂	1	0	0.6±0.09	1	0.4	—	24.9	6	14±1.7	13	0	4±1.2	6.8	2	3.7±0.5
V ₁₃	0.2	0.2	0.2±0.09	0	0	0	26	6	15±1.5	7	0	4.4±0.6	8.5	4	5.5±0.4
V ₁₄	0.2	0.2	0.2±0.09	0	0	0	24.7	5	13±1.5	5	0	2±0.64	6.8	1.8	3.4±0.43
V ₁₅	0.2	0.2	0.2±0.09	1	0.3	0.25±0.1	23	5	14±1.5	5	0	1.2±0.2	6	2	4±0.3
V ₁₆	0.2	0.2	0.2±0.09	1.2	0	0.5±0.11	24.5	3	14±1.5	4	0	—	3.9	1	2.7±0.8
V ₁₇	0.2	0.2	0.2±0.09	1	0	0.3±0.10	12.5	3	10±0.6	4	0	0.6±0.2	3.5	2	3±0.2

Table 13

Lead	Time of appearance of internal deviation in sec.			Tap	Time of appearance of internal deviation in sec.		
	Max.	Min.	M ±		Max.	Min.	M ±
V ₁	0.021 0.020	0.010 0.010	0.016±0.001 0.010±0.0003	V ₁	0.045 0.040	0.020 0.020	0.038±0.002 0.030±0.001
V ₂	0.030 0.020	0.010 0.010	0.020±0.001 0.020±0.0002	V ₂	0.040 0.040	0.020 0.020	0.034±0.002 0.030±0.002

2. Vector Cardiography

Table 14

* loop QRS in degrees	Sect. of arr. of loop QRS in degrees			Angle QRS-T in degrees			Area of loop QRS in cm.			Max Val. of loop QRS vec. in cm.					
	Max. Min.		M ±	Max. Min.		M ±	Max. Min.		M ±	Max. Min.		M ±			
	Max.	Min.		Max.	Min.		Max.	Min.		Max.	Min.				
I	+137 +113	+56 +71	+94±4.3 +93±3	+139 +105	+59 +62	+93±6.08 +91±3	37 26	3 1	13±2.75 13±2.6	1.1 0.9	0.4 0.3	0.66±0.059 0.50±0.04	2.7 2.2	0.8 0.9	1.58±0.52 1.6±0.359
II	+93 +101	+43 +73	+77±3.7 +85±2	+113 +116	+71 +76	+92±4.3 +93±3.2	42 40	3 3	16±3.06 16±3	1.1 1.4	0.4 0.4	0.70±0.056 0.70±0.06	2.7 2.2	0.7 1.1	1.57±0.407 1.53±0.63
III	+117 +107	+23 +62	+85±6.14 +86±2.8	+112 +110	+33 +49	+77±5.5 +77±4	28 32	1 0	12±2 14±2.2	1.7 1.2	0.4 0.4	0.75±0.094 0.60±0.05	2.3 2.2	0.8 0.7	1.5±0.46 1.53±0.39
IV	-170 -110	-76 -62	-110±8.04 -92±3.5	-174 -111	-66 -73	-108±8.74 -96±3.2	33 46	2 1	14±2.8 11±2.8	0.8 0.9	0.2 0.1	0.45±0.05 0.40±0.04	2.1 1.9	0.8 0.5	1.24±0.40 1.13±0.39
V	-175 -142	-75 -52	-116±10.12 -108±8.5	-153 -139	-63 -72	-195±7.24 -114±4.5	42 41	1 3	11±3.47 11±2.8	1.05 0.9	0.2 0.2	0.40±0.09 0.30±0.025	1.7 1.0	0.6 0.4	1.0±0.125 0.66±0.18

*According to Akul'shev

3. Spatial Vectorcardiography^{1/}

Table 15

Position of QRS vector on the frontal plane in degrees			Position of vector T on the frontal plane in degrees			Angle between vectors QRS and T in degrees			Position of vector QRS on the horiz. plane in degrees			Position of vector T on the horiz. plane in degrees		
Max.	Min.	M + σ	Max.	Min.	M + σ	Max.	Min.	M + σ	Max.	Min.	M + σ	Max.	Min.	M + σ
Right downwd +109	left dnwd +21	left downwd +67 \pm 6,9	left downwd +87	left dnwd +29	left downwd +49 \pm 4,5	47	3	26 \pm 4,5	Backward 50	Forward 20	Backward 24 \pm 4,54	Backward 30	Forward 15	Backward 2,5 \pm 3,3
Right downwd +82	left dnwd +3	left downwd +46 \pm 7	left downwd +87	left dnwd +7	left downwd +48 \pm 6	57	0	24 \pm 5	Backward 40	Forward 10	Backward 29 \pm 2	Backward 0	Forward 15	Backward 0

^{1/}Position of vector QRS or T on the horizontal plane is calculated on the basis of diversion of this vector, from the frontal plane passing through the electrical center of the heart.

B. MECHANICAL HEART ACTIVITY

1. Ballistocardiography

Table 16

Values	Time intervals in seconds											
	R-H	R-I	R-J	R-K	H-K	I-K	H-I	I-J	J-K	K-L		
Max.	0,12 0,12	0,20 0,18	0,30 0,26	0,40 0,35	0,32 0,26	0,22 0,20	0,10 0,07	0,11 0,09	0,11 0,14	0,13 0,15		
Min.	0,08 0,06	0,10 0,10	0,20 0,19	0,31 0,27	0,22 0,20	0,17 0,14	0,04 0,03	0,06 0,07	0,06 0,07	0,06 0,07		
M + σ	0,09 \pm 0,013 0,08 \pm 0,005	0,17 \pm 0,086 0,14 \pm 0,006	0,26 \pm 0,004 0,22 \pm 0,004	0,36 \pm 0,033 0,32 \pm 0,005	0,26 \pm 0,008 0,23 \pm 0,006	0,19 \pm 0,005 0,18 \pm 0,005	0,08 \pm 0,006 0,06 \pm 0,003	0,10 \pm 0,004 0,08 \pm 0,003	0,09 \pm 0,004 0,09 \pm 0,004	0,10 \pm 0,006 0,09 \pm 0,004		

Table 17

Values	Wave Amplitudes in mm.				Coefficients			Respira- tory co- efficient	Ballisto- cardio- graphic coef.	Intra- systolic indicator in %
	HI	IJ	JK	KL	HI/II	JK/IJ	KL/IJ			
Max.	16 11	23 17	22 20	21 14	0.72 0.57	1.40 1.50	1.20 1	2.4 3.4	0.71 0.68	75 78
Min.	5 3	10 9	11 8	5 4	0.20 0.28	0.92 0.80	0.50 0.28	1.3 1.3	0.23 0.30	61 62
M ± σ	10 ± 1.15 6 ± 6	16 ± 1.27 13.5 ± 0.3	17 ± 1.39 14 ± 1.09	14 ± 1.7 9 ± 8	0.57 ± 0.052 0.50 ± 0.04	1.07 ± 0.049 1 ± 0.05	0.86 ± 0.09 0.70 ± 0.05	1.83 ± 0.1 2.1 ± 0.2	0.47 ± 0.033 0.45 ± 0.003	70 ± 1.43 73 ± 1.5

2. Phonocardiography

Table 18

Values	Duration of the I sound (sec.)				Duration of the II sound (sec.)				Ratio of Ampil- tudes I/II	
	Ha	A	T	Pa	Ha	A	T	Pa	Ha	A
Max.	0.19 0.12	0.18 0.11	0.14 0.12	0.13 0.09	0.13 0.10	0.11 0.10	0.12 0.10	0.12 0.10	1.23 2.30	1 1.16
Min.	0.10 0.10	0.08 0.05	0.10 0.08	0.09 0.05	0.08 0.04	0.08 0.05	0.09 0.05	0.09 0.05	0.35 0.42	0.25 0.45
M ± σ	0.12 ± 0.005 0.11 ± 0.002	0.11 ± 0.007 0.08 ± 0.003	0.12 ± 0.004 0.09 ± 0.003	0.11 ± 0.003 0.08 ± 0.004	0.10 ± 0.004 0.07 ± 0.004	0.10 ± 0.002 0.07 ± 0.003	0.10 ± 0.002 0.07 ± 0.003	0.10 ± 0.002 0.07 ± 0.004	0.91 ± 0.243 1.45 ± 0.16	0.69 ± 0.073 0.66 ± 0.080

1/ Ha - heart sound, A - aortic valve, T - tricuspidal valve, P.A. - pulmonary artery

3. Combined Polycardiographic Investigation Table 19

Values	Duration of ventricular contraction in sec.					
	Tensile Phase	Trans-formation period	Period of increased pressure	Ejection phase	Mechanical systole	Electrical systole
Max.	0.10 0.10	0.08 0.07	0.05 0.05	0.32 0.33	0.36 0.36	0.42 0.39
Min.	0.05 0.06	0.04 0.03	0.01 0.01	0.22 0.25	0.27 0.28	0.30 0.34
M ± σ	0.08 ± 0.004 0.08 ± 0.003	0.06 ± 0.004 0.05 ± 0.002	0.02 ± 0.004 0.03 ± 0.002	0.28 ± 0.007 0.29 ± 0.005	0.31 ± 0.007 0.32 ± 0.005	0.36 ± 0.010 0.36 ± 0.000
						0.41 0.41 0.33 0.33 0.36 ± 0.007 0.37 ± 0.006

Table 20

Values	Internal coefficient of systole (ISC)	Internal Tensile coefficient (ITC)	Corrected indicator of tension of (CIT) in %	Corrected indicator of eject. in % (MED)	Mechan. elect. indicator
Max.	0.50 0.35	6 6	31 25	97 97	97 97
Min.	0.11 0.21	1.25 0.80	15 16	82 84	64 82
M ± σ	0.28 ± 0.024 0.27 ± 0.010	3.54 ± 0.6 2.30 ± 0.3	22 ± 1.32 21 ± 1.3	91 ± 1.12 89 ± 1.0	85 ± 2.84 89 ± 1.5

From our observations of many patients with various diseases of the cardiovascular system we took notice of the considerable advantages offered by synthetic electrocardiology as compared with the analytical study of the data obtained individually for each method of investigation. Such an approach not only aids in the determination and refinement of functional and anatomical diagnosis, but also challenges every physician-cardiologist to think more deeply for explanations of the mechanisms of observed deviations, to draw clinical conclusions or to create new theoretical hypotheses and to seek new methods for further scientific research.

In the exposition of this book we have confined ourselves to pathophysiological principles and rarely reported the results of individual nosological forms. We have mentioned certain of them only as examples. We certainly do not deny characteristic peculiarities in the electrocardiologic deviations of various pathological forms, and in further studies we intend to present the patterns of synthetic electrocardiology for individual nosological units. With the use of the data of this book, however, any clinician can achieve a broad understanding of the electrocardiologic pattern of a certain definite disease. Therefore, from the pathophysiological approach of this book he can arrive at anatomical conclusions, because he now knows which cardiac functions are impaired by the disease under consideration, which section becomes hypertrophied, how the blood supply is affected, etc.

In concluding this book we wish to emphasize certain facts. As has been stated in the introduction, while submitting the principle of synthetic electrocardiology and exploring the problem of organizing the laboratory and training its staff, we have never denied the importance of the work performed by specialists in the somewhat narrower areas of electrocardiography, vectorcardiography, ballistocardiography or phonocardiography. The work of these specialists is of great value, and all further development of the methodology of synthetic electrocardiology depends on their findings and achievements.

We think that there is a necessity for further improvement in the methodology of synthetic electrocardiology. This may be accomplished by enriching the number of diagnostic criteria of the methods described in this book. It is also necessary to develop new methods for investigation of the electrical and mechanical activity separately for the right and the left halves of the heart; there is need for the further study of blood flow into individual heart chambers and for development of new functional indicators, etc. A special effort must be made in the study of the diastolic period of cardiac contraction. We find that the diastole is a very active physiological process and that it should have its physical manifestations. In the exposition of this 367 book we discussed primarily the systole of the heart. We by-passed the diastole because as yet there are no successful methods for the study of this important part of cardiac activity.

In addition we feel the necessity of using the methodology of synthetic electrocardiology for the study of the following important theoretical and clinical questions: how insufficiencies of the left and right ventricles

affect electrical and mechanical cardiac activity and how various diseases cause insufficiency of either of these two types. How these processes progress and whether these processes have a common or different mechanism of development.

For the solution of these problems it is necessary to undertake further explorations and to apply the latest developments of clinical and experimental electrophysiology and biophysics. These are some of the problems facing the contemporary electrocardiologist. If he wants to become an artist in this important and noble undertaking he must explore because, "to know how to investigate is the greatest part of science." (Hippocrates)

Modern cardiology has access to a large number of methods for the investigation of cardiac action. A very important place among them belongs to electrocardiography, the basis for all knowledge concerning cardiac function, the corner-stone of modern cardiological science. During recent years we have witnessed continued progress in this field, especially in the use of new electrocardiographic techniques and in applying the vectorial principles of interpretation of different electrophysiological manifestations.

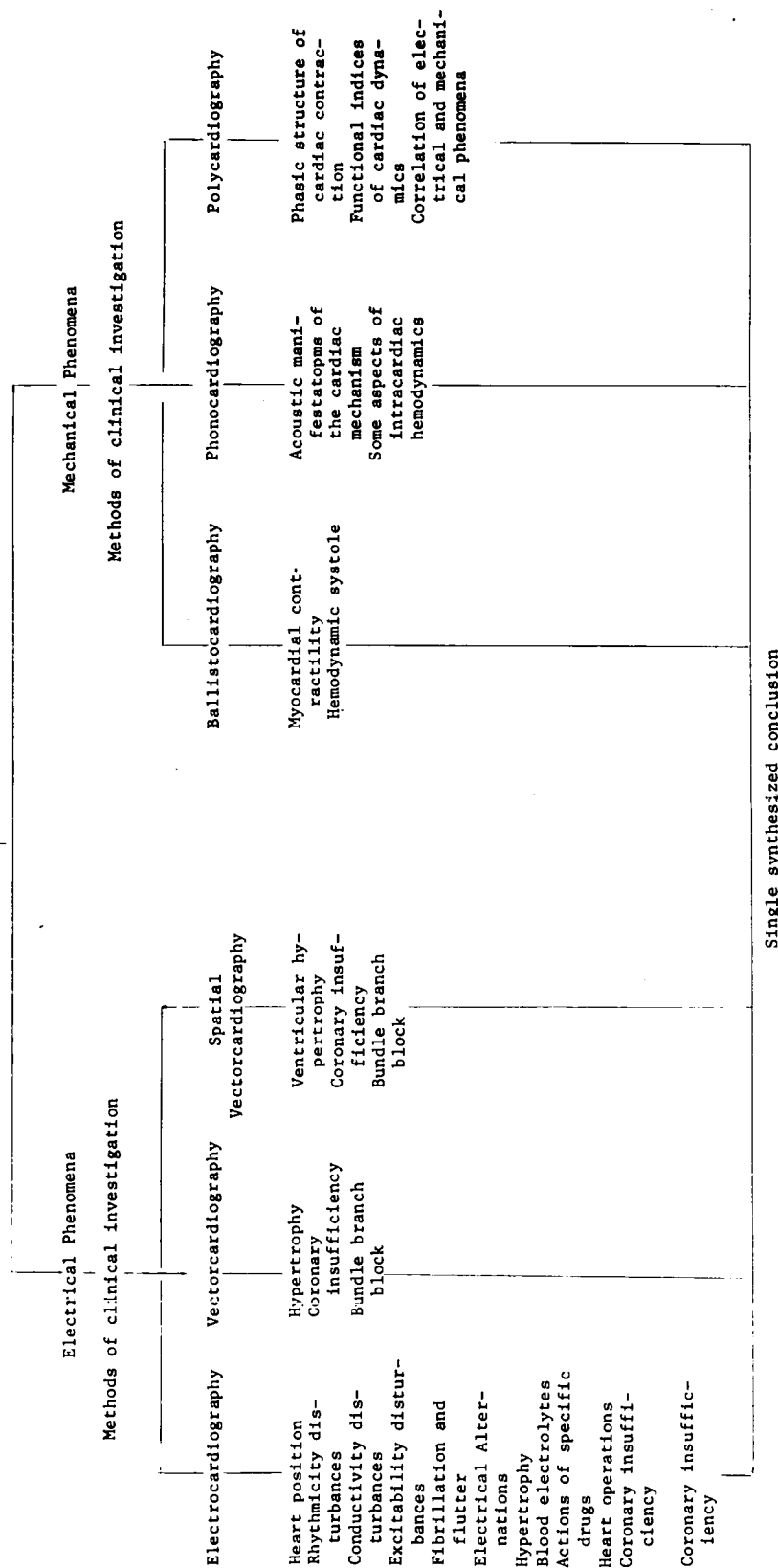
The modern electrocardiologist, however, cannot limit himself to studying only the electrical activity of the heart. In order to obtain a thorough and comprehensive picture of cardiac action it is necessary also to study its mechanical activity and to fill up the gaps of electrocardiography. It must be quite clear that the latter can provide only very poor, and even then indirect, information on the most important property of the heart muscle - its contractility.

At the Department of Electrocardiology, Institute of Cardiology and Cardiac Surgery, Academy of Sciences, Armenian SSR, such methods as electrocardiography, ballistocardiography, phonocardiography and combined polycardiographic investigation are widely used in routine practice, as well as in clinical research or experimental investigations. In carrying out this work we have repeatedly met with the fact that each of these methods has definite limits, being able to elucidate only a particular aspect of cardiac action and answer only a specific group of questions. Based on our experience, which includes a great number of observations in most of the fields of modern cardiology and cardiac surgery, we have come to the firm conviction that it is necessary to tackle these methods from one common viewpoint and carry out a general and full synthesis of the analytical data provided by each of them. Thus, all these separate methods are unified into a synthetic entity. Such an approach considerably increases the significance and specific contribution of electrocardiology in the complex of cardiological methods.

We have named this trend in modern clinical electrophysiology and biophysics of the heart "Synthetic Electrocardiology." All the above-mentioned methods have been united under the term electrocardiology not, of course, on the basis of the etymology of that word (that would have been theoretically wrong), but on the basis of the essence of that term, as we understand it today, and its modern content.

The book consists of four parts. The first part is devoted to the bases of synthetic electrocardiology. Complex biochemical and biophysical processes underlie the heart beat; these processes, unfortunately, cannot yet be made the object of routine clinical investigation, but as a final result of cardiac contraction, electrical and mechanical phenomena develop which, being physical processes, open up vast possibilities for large-scale investigations; this is not at all difficult today when the enormous achievements of radioelectronics have made it possible to design advanced apparatus and devices which can supply the investigator with inestimable information of paramount importance. In

CONTRACTION OF THE HEART



Single synthesized conclusion

this part, brief anatomical and physiological data are presented, as well as some information on the electrophysiology of the heart and the mechanism of cardiac contraction. The methodology of synthetic electrocardiology is presented in schematic form (see the preceding page).

The second part is called "Clinical Study of the Electrical Activity of the Heart." It consists of three chapters: Electrocardiography, Vectorcardiography and Spatial Vector Electrocardiography. In each chapter full details are presented on all the theoretical and practical aspects of the corresponding method; all categories and criteria are described from the standpoint of synthetic methodology.

The third part "Clinical Study of the Mechanical Activity of the Heart" has the same construction. Three chapters are discussed here from an analogous point of view: Ballistocardiography, Phonocardiography and Combined Polycardiographic Investigation.

The fourth part is devoted to the practical questions of synthetic electrocardiology. A diagram is presented showing the significance and the role which the above-mentioned methods must play in studying the four physiological properties of the heart and the principal pathological syndromes. At the end of this part an example is given of the electrocardiologic report form; tables of the principal findings of all these methods as determined in two age groups of normal persons are presented here.

The book ends with a list of references.

This monograph is presented against a pathophysiological background; no nosological forms are discussed, except in a few cases. We believe that in each individual case the specialist, recognizing the various electrophysiological and biophysical patterns and basing himself on clinical and other data, can come to a definite pathoanatomical conclusion.

While developing the principles of synthetic electrocardiology and proposing them for large-scale clinical use, we at the same time do not deny the role of particular specialists in each of the methods mentioned above. Such specialists, especially research workers, are needed for each narrow field of modern electrocardiology; further enrichment of the synthetic method depends on their work. The development of our science requires the joint efforts of clinicians, physiologists, biophysicists, electronic engineers and experts in other fields of science. This is particularly important in our time when the methods of cybernetics and electronic computers are more and more widely used in medicine, and when telemetric techniques are needed for human space flights.

Zaven L. Dolabchyan

Yerevan, 1963

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